HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

NOT TO CIRCULATE

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

NOT TO CIRCULATE

Digitized by the Internet Archive
in 2015

https://archive.org/details/connecticutmedic5716conn

Connecticut Medicine

Vol. 57, No. 1 *The Journal of the Connecticut State Medical Society* January 1993
160 St. Ronan Street, New Haven, CT 06511 (203) 865-0587

CSMS OFFICERS
Sultan Ahamed, M.D., Norwich
President
Howard J. Wetstone, M.D., Hartford
President-Elect
Theodore Zanker, M.D., New Haven
Vice-President
Marjorie G. Petro, M.D., Willimantic
Secretary
Robert R. McDonnell, M.D., New Haven
Treasurer
Michael M. Deren, M.D., New London
Speaker of the House of Delegates
Neil H. Brooks, M.D., Rockville
Vice-Speaker of the House of Delegates

AMA DELEGATES
Jerome K. Freedman, M.D., Branford,
Neil H. Brooks, M.D., Rockville, *Alternate*
Joseph S. Sadowski, M.D., Hartford,
James P. Orphanos, M.D., Greenwich, *Alternate*
Stewart J. Petrie, M.D., Woodbridge,
John B. Franklin, M.D., Hartford, *Alternate*
Edward A. Kamens, M.D., Fairfield,
Joseph C. Czarsty, M.D., Oakville, *Alternate*
Jerome Bobruff, M.D., New London,
Roger S. Beck, M.D., Wethersfield, *Alternate*

COUNCILORS
Dickerman Hollister, Jr., M.D., Greenwich
Fairfield County
Stanley J. Keating, M.D., Simsbury
Hartford County
Howard W. Meridy, M.D., Torrington
Litchfield County
Juan B. Sosa, M.D., Middletown
Middlesex County
Steven Wolfson, M.D., New Haven
New Haven County
Joseph P. Zeppieri, M.D., New London
New London County
Gerald L. Schwartz, M.D., Rockville
Tolland County
Stephen J. Leach, M.D., Storrs
Windham County

ASSOCIATE COUNCILORS
Lawrence M. Blum, M.D., Fairfield
Fairfield County
Robert D. Cottle, M.D., Stamford
Fairfield County
Brendan I. Fox, M.D., Hartford
Hartford County
Wayne F. Hosking, M.D., Hartford
Hartford County
Michael R. Tesoro, M.D., Hartford
Litchfield County
Donald D. Timmerman, M.D., Glastonbury
Middlesex County
David W. Parke, M.D., Meriden
New Haven County
Ellen R. Fischbein, M.D., Cheshire
New Haven County
Thomas H. Lesnik, M.D., Norwich
New London County
Francis Van Nostrand, M.D., Stafford Springs
Tolland County

COUNCILOR AT LARGE
James P. Orphanos, M.D., Greenwich

Articles, Editorials, and **Advertisements,** published in *Connecticut Medicine* do not necessarily represent the official position or endorsement of *The Journal of the Connecticut State Medical Society,* or the Connecticut State Medical Society itself. The *Journal* reserves the right to make the final decision on all content and advertisements.

Editor	Robert U. Massey, M.D.
Literary Editor	Sherwin B. Nuland, M.D.
Editor Emeritus	J. Alfred Fabro, M.D.
Managing Editor	Wanda E. Jacques
Editorial Assistant	Janet Massey

Editorial Board

Orthopedic Surgery	William R. Cambridge, M.D.
General Surgery	H. David Crombie, M.D.
General Practice	Evans H. Daniels, M.D.
Cardiology and Internal Medicine	Martin Duke, M.D.
Psychiatry	Stephen Fleck, M.D.
Dermatology	Robert D. Greenberg, M.D.
Nephrology and Internal Medicine	William A. Handelman, M.D.
Radiology	E. Marvin Henkin, M.D.
Pathology and Hematology	Ramon N. Kranwinkel, M.D.
Neurology	Martin W. Kremenitzer, M.D.
Pediatrics	Julius Landwirth, M.D.
Orthopedic Surgery	Frank W. Maletz, M.D.
General Surgery	Sherwin B. Nuland, M.D.
Pathology	John L. Meyer, M.D.
Ophthalmology	David W. Parke, M.D.
Obstetrics and Gynecology	Stewart J. Petrie, M.D.
Anesthesiology	James J. Richter, M.D.
Neurosurgery	Melville P. Roberts, M.D.
Cardiology	Michael A. Rossi, M.D.
Internal Medicine and Rheumatology	Robert T. Schoen, M.D.
Family Practice	Robert K. Shapter, M.D.
Emergency Medicine	Vijay K. Sikand, M.D.
Internal Medicine and Geriatrics	James E.C. Walker
Orthopedic Surgery	Ulrich H. Weil, M.D.

CSMS Staff

Executive Director	Timothy B. Norbeck
Associate Executive Director	Josephine P. Lindquist
Director of Scientific Activities	Robert J. Brunell
Director of Public Affairs	Marc J. Garofalo
Director of Government Relations	Kyle L. Ballou, Esq.
Director of Special Services	Margaret K. Connelly
Membership Department	Dorothy M. Stratton
Placement Services	Pina Morgillo
Management Information Systems	Deborah P. Maynes
Controller	Marc A. Sullivan
Administrative Assistant	Patricia Mastriano
Administrative Assistant	Stefanie Hobbs

HEALTH SCIENCES LIBRARY

For Your Patients with Minor to Moderate Burns:

Bridgeport Hospital's Burn Wound Program

For three decades, Bridgeport Hospital's Burn Center has provided sophisticated inpatient care for burn patients. Our Burn Team includes physicians, nurses, therapists, dietitians, and social workers with special training in the care of burn patients.

Now this team of experts offers you and your patients daily specialized outpatient care for minor and moderate burns as classified by the American Burn Association's Injury Severity Grading System.

Patients are accepted *only by referral from their primary physician or an Emergency Department physician.* You will receive a complete initial evaluation of your patient's condition, and follow-up reports as indicated.

Patients who qualify for referral to the Burn Wound Program will meet the following criteria:

Magnitude of Burn Injury (% TBSA)	Second Degree Burn Adult	Children (% TBSA)	Third Degree (% TBSA)	Special Location	Electrical Inhalation Poor Risk Trauma
Major	>25%	>20%	>10%	—	—
Moderate	15–25%	10–20%	2–10%	—	—
Minor	5–15%	5–10%	1–2%	—	—

For more information, for referrals, or to consult with a physician who specializes in burn care, call **Michael L. D'Aiuto, MD,** director, Bridgeport Hospital Burn Center, at **384-3728.**

BRIDGEPORT
HOSPITAL

Comprehensive Healthcare
For Southern Connecticut

Connecticut Medicine

The Journal of the Connecticut State Medical Society

VOLUME 57 JANUARY 1993 NUMBER 1

STACKS

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

MAR 2 1993

REC'D NOT IN CIRC.

scientific:

Charles W. Needham 3 Pinched Nerves and Signature Signs
Michael N. Kessler, Lawrence G. Raisz, and Marja M. Hurley 9 Interactive Effects of Basic Fibroblast Growth Factor and Heparin on Bone in 21-Day Fetal Rat Calvariae
Vincent L. Perez and Peter M. Shimkin 13 Computed Tomography in Pulmonary Tuberous Sclerosis
Alex A. Cardoni 15 Psychopharmacology of Nicotine: Implications for Pharmacotherapy in Psychiatry

special articles:

Patrick J. Carolan and Vincent J. Catrini 19 The Choice Is Ours
Dickerman Hollister, Jr. 25 Improving Health-care Costs with Minimal Compromise
Tsuyoshi Inoshita 27 The Education of a Pathologist
Drug Information Update: Hartford Hospital
Elizabeth Chime-Udeh 29 Stress-Related Gastritis: Focus on the Use of H_2-receptor Antagonists and Antacids
Epitomes of Progress 33 Important Advances in Clinical Medicine—Radiology

STACKS

departments:

Fifty Years Ago
43 The Beveridge Plan
The President's Page
Sultan Ahamed 45 Cost of Health Care—The Truth
Reflections on Medicine
Robert U. Massey 47 Real Reform or Just Tinkering?
Law, Medicine, and Public Policy
Joseph M. Healey 49 Ends and Means in Organ Transplantation: The Anencephalic Donor
Letters to the Editor
Edward J. Volpintesta 51 Defensive Medicine
Sidney Vernon 51 Cooperation and Compromise
Medical News Capsules
Timothy B. Norbeck 52 This Month's Reading in Review

STACKS

Health Sciences Library 7
University of Maryland
111 South Greene Street
Baltimore MD 21201

general:

53 Book Reviews
54 CSMS Physician Placement Service
56 Information for Authors

Owned, Published and Copyrighted, © 1993 by the Connecticut State Medical Society

CONNECTICUT MEDICINE (ISSN 0010-6178), published monthly at 160 St. Ronan St., New Haven, CT 06511
Subscription rate included in membership dues. Non-members: $25.00 per year. Second-class postage paid at New Haven, CT
POSTMASTER: Send address changes to 160 St. Ronan St., New Haven, CT 06511

Connecticut Medicine

The Journal of the Connecticut State Medical Society

VOLUME 57 FEBRUARY 1993 NUMBER 2

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

STACKS

MAR 18 1993

REC'D NOT IN CIRC.

scientific:

Anthony P. Polednak and John T. Flannery 59 Trends in Breast Cancer Treatment in Connecticut

Elaine E. Yordan and Robert A. Yordan 65 The Maternity Home for Adolescents: A Concept from the Past Fulfilling a Contemporary Need

Stewart P. Seigle 69 On Death and Dying

Epitomes of Progress 73 Important Advances in Clinical Medicine—Chest Diseases

The Medical Letter 85 Drugs for Pain

special articles:

Martin Duke 91 An Historical Review of Rupture of the Heart

Peter V. Tishler 97 "The Care of the Patient": A Living Testimony to Francis Weld Peabody

Centers for Disease Control 103 Deaths and Hospitalizations from Chronic Liver Disease and Cirrhosis—United States, 1980-1989

editorial:

Stewart J. Petrie 107 An Outgoing, Delegate's Point of View

departments:

Fifty Years Ago

109 The Spa in America

The President's Page

Sultan Ahamed 111 Biomedical Research—Our Moral Responsibility

Reflections on Medicine

Robert U. Massey 113 Managed Competition: The Rogue Wave of the Future?

Letters to the Editor

Edward J. Volpintesta 114 Malpractice *vs* Humanism

Sidney Vernon 114 Self-rape is Partial Suicide

Steven H. Stein 114 RBRVS in Rural Connecticut

Steven Wolfson 115 Birth of a New Health Care System

Medical News Capsules

Timothy B. Norbeck 116 This Month's Reading in Review

general:

117 Book Review

118 CSMS Physician Placement Service

120 Information for Authors

STACKS

STACKS

Health Sciences Library 7
University of Maryland
111 South Greene Street
Baltimore MD 21201

Owned, Published and Copyrighted, © 1993 by the Connecticut State Medical Society

CONNECTICUT MEDICINE (ISSN 0010-6178), published monthly at 160 St. Ronan St., New Haven, CT 06511
Subscription rate included in membership dues. Non-members: $25.00 per year. Second-class postage paid at New Haven, CT
POSTMASTER: Send address changes to 160 St. Ronan St., New Haven, CT 06511

Connecticut Medicine

The Journal of the Connecticut State Medical Society

OLUME 57 MARCH 1993 NUMBER 3

STACKS

scientific:

Ross I.S. Zbar, William B. Crede, Charles F. McKhann, and James F. Jekel 123 **The Postoperative Incidence of Small Bowel Obstruction Following Standard, Open Appendectomy and Cholecystectomy: A Six-Year Retrospective Cohort Study at Yale-New Haven Hospital**

J.P. Mulhall and A.D. Drezner 129 **Postoperative Compartment Syndrome and the Lithotomy Position: A Report of Three Cases and Analysis of Potential Risk Factors**

Daniel T. Engelman, John Thayer, John Derossi, Jacob Scheinerman, and Louis Brown 135 **Pregnancy Related Coronary Artery Dissection: A Case Report and Collective Review**

Newington Children's Hospital 140 **Abstracts: 30th Annual Orthopaedic Conference**

Epitomes of Progress 147 **Important Advances in Clinical Medicine—Ophthalmology**

special articles:

Faith McGrath and Nicholas G. Tomaiuolo 155 **Practice Locally, Search Globally**

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

APR 6 1993

REC'D. NOT IN CIRC.

STACKS

STACKS

Health Sciences Library 7
University of Maryland
111 South Greene Street
Baltimore MD 21201

departments:

Fifty Years Ago

G.M. Mackenzie 163 **Social Functions of the Modern Hospital**

The President's Page

Sultan Ahamed 167 **Ivory Towers and Organized Medicine**

Law, Medicine, and Public Policy

Joseph M. Healey 169 **The Anencephalic as Organ Donor: Issues at the Beginning and End of Life**

Reflections on Medicine

Robert U. Massey 171 **Listening to the Patient, Primary Care, and Reform**

Letters to the Editor

Sidney Vernon 173 **Light at the End of the Tunnel of Time**

Edward J. Volpintesta 173 **Fear of Malpractice Litigation**

Claude A. Frazier 173 **Looking for Reports of Anaphylaxis**

Medical News Capsules

Timothy B. Norbeck 174 **This Month's Reading in Review**

CSMS Council 175 **From the Executive Director's Office**

general:

180 **CSMS Physician Placement Service**

182 **Information for Authors**

Owned, Published and Copyrighted, © 1993 by the Connecticut State Medical Society

---

CONNECTICUT MEDICINE (ISSN 0010-6178), published monthly at 160 St. Ronan St., New Haven, CT 06511
Subscription rate included in membership dues. Non-members: $25.00 per year. Second-class postage paid at New Haven, CT
POSTMASTER: Send address changes to 160 St. Ronan St., New Haven, CT 06511

Connecticut Medicine

The Journal of the Connecticut State Medical Society

VOLUME 57 APRIL 1993 NUMBER 4

scientific:

John D. Thompson, Keon-Hyung Lee, John T. Lynch, and Donna Diers 187 "Where Have All the Children Gone"? Inpatient Pediatric Care in Connecticut

Michael C. Lindberg, David Winsemius, Bernadine Lawer, and Roy V. Erickson 197 Tuberculin Reactivity Is Low Among Residents of a Connecticut Nursing Home

Epitomes of Progress 201 Important Advances in Clinical Medicine—Anesthesiology

special articles:

W. Raymond James 211 The Connecticut State Medical Society and Malpractice The First 180 Years

American College of Physicians, National Commission on Correctional Health Care, and American Correctional Health Services Association 224 The Crisis in Correctional Health Care: The Impact of the National Drug Control Strategy on Correctional Health Services

Drug Information Update: Hartford Hospital

Heather Kertland 232 Nitrate Tolerance—Mechanisms and Prevention

editorial:

Timothy B. Norbeck 235 Rising Health Care Costs: Disease or Symptom?

departments:

Fifty Years Ago

239 Medicine and the War

The President's Page

Sultan Ahamed 241 Medicare Mess

Law, Medicine, and Public Policy

Joseph M. Healey 243 Doctors, Patients, and the End of Life

Reflections on Medicine

Robert U. Massey 245 Health-Care Reform and Long-Term Care

Letters to the Editor

Sidney Vernon 247 Sudden Cardiac Death

Edward J. Volpintesta 247 In Response to Dr. Ahamed

Norman M. Canter 247 Keep the Primary Care Physician in the Hospital

W. David Zitzkat 248 "Attractive Educational Brochures"

Victor A. Altshul 249 In Response to "Real Reform or Just Tinkering?"

Medical News Capsules

Timothy B. Norbeck 251 This Month's Reading in Review

CSMS Council 253 From the Executive Director's Office

general:

249 CSMS Milestones—1792-1992

259 In Memoriam

260 CSMS Physician Placement Service

263 Information for Authors

STACKS

STACKS

STACKS

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

MAY 20 1993

REC'D. NOT IN CIRC.

Health Sciences Library
University of Maryland
111 South Greene Street

Owned, Published and Copyrighted, © 1993 by the Connecticut State Medical Society

---

CONNECTICUT MEDICINE (ISSN 0010-6178), published monthly at 160 St. Ronan St., New Haven, CT 06511
Subscription rate included in membership dues. Non-members: $25.00 per year. Second-class postage paid at New Haven, CT
POSTMASTER: Send address changes to 160 St. Ronan St., New Haven, CT 06511

Connecticut Medicine

The Journal of the Connecticut State Medical Society

VOLUME 57 MAY 1993 NUMBER 5

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

JUN 11 1993

REC'D. NOT IN CIRC.

STACKS

STACKS

Health Sciences Library 7
University of Maryland
111 South Greene Street
Baltimore MD 21201

Special Issue: Geriatrics

James E.C. Walker, M.D., Guest Editor

scientific:

James E.C. Walker 267 Aging—A Not So Risky Affair
James O. Judge 269 Exercise Programs for Older Persons: Writing an Exercise Prescription
Richard A. Marottoli 277 Driving Safety in Elderly Individuals
Leo M. Cooney, Jr. 281 Assessment of Immobility in the Elderly
Richard W. Besdine 287 Stroke Prevention in the Elderly
James E.C. Walker 293 Congestive Heart Failure in the Elderly
Fitzhugh C. Pannill, III 299 Urinary Incontinence for the Primary Care Physician
Sharon K. Inouye 309 Delirium in Hospitalized Elderly Patients: Recognition, Evaluation, and Management
Henry Schneiderman 317 Physical Examination of the Aged Patient

special articles:

Peter J. Deckers 325 What Matters Most!
W. Raymond James 331 The Connecticut State Medical Society and Professional Liability Insurance—1971 to 1983

editorial:

Paul Dolinsky 339 Preventive Medicine for Senior Citizens: Uncovered
R.M. Fasanella 341 Age-Related Macular Changes: A Plea
John F. Raycroft 342 OREF

departments:

Fifty Years Ago
Howard W. Haggard 345 Connecticut Colonial Medicine
The President's Page
Sultan Ahamed 349 Doctor's Worth
Law, Medicine, and Public Policy
Joseph M. Healey 351 The Emerging Legal Consensus About End of Life Decisions
Reflections on Medicine
Robert U. Massey 353 Geriatrics: Defining a New Specialty
Medical News Capsules
Timothy B. Norbeck 355 This Month's Reading in Review

general:

357 Information for Authors
358 CSMS Physician Placement Service

STACKS

Connecticut Medicine

The Journal of the Connecticut State Medical Society

VOLUME 57 JUNE 1993 NUMBER 6

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

STACKS

JUL 14 1993

REC'D. NOT IN CIRC.

Health Sciences Library 7
University of Maryland
111 South Greene Street
Baltimore MD 21201

scientific:

Linda M. Preble, Lawrence Perlstein, Lori Katsoff-Seidman, Theresa Z. O'Connor, and Paul G. Barash 363 The Patient Care Evaluation System: Patients' Perception of Anesthetic Care
Albert N. Browne-Mayers 367 Introducing Physicians to Case Management in Psychiatry
Allen L. Hixon 373 Social Correlates of Malnutrition Among Filipino Street Children
Epitomes of Progress 377 Important Advances in Clinical Medicine—Neurosurgery
The Medical Letter 391 Choice of Cholesterol-lowering Drugs

special articles:

W. Raymond James 395 The Connecticut State Medical Society and Connecticut Medical Insurance Company
Derrik F. Woodbury 405 A Doctor's Child Is Born
Russell V. Fuldner 409 Home to Carthage

departments:

Fifty Years Ago

Alice Hamilton 411 The International Control of Disease After the War
Yale University 415 Problems of International Health

The President's Page

Howard J. Wetstone 419 The New President's Message

STACKS

Law, Medicine, and Public Policy

Joseph M. Healey 420 Reexamining Liability for Acts that Lead to Death

Reflections on Medicine

Robert U. Massey 421 I Swear by Apollo Physician

Medical News Capsules

Timothy B. Norbeck 422 This Month's Reading in Review

Letters to the Editor

Margaret L. Pearson 423 Medical Hydrology
Harvey N. Mandell 423 In Response to Dr. Volpintesta

From the Executive Director's Office

CSMS 425 Council Minutes
CSMS 429 Annual Meeting of the House of Delegates
Sultan Ahamed 433 Address of the Retiring President
Timothy B. Norbeck 435 Address of the Executive Director

STACKS

general:

440 CSMS Physician Placement Service

Owned, Published and Copyrighted, ©1993 by the Conntecticut State Medical Society

CONNECTICUT MEDICINE (ISSN 0010-6178), published monthly at 160 St. Ronan St., New Haven, CT 06511
Subscription rate included in membership dues. Non-members: $25.00 per year. Second-class postage paid at New Haven, CT
POSTMASTER; Send address changes to 160 St. Ronan St., New Haven, CT 06511

Pinched Nerves and Signature Signs

CHARLES W. NEEDHAM, M.D.

HARVEY Cushing called bitemporal hemianopia the signature of the optic chiasm. Bitemporal loss of visual fields, like a horse wearing blinders, points nowhere else in the nervous system. Only in the chiasm do optic nerve fibers from the nasal half of each retina cross in the midline, so that temporal field loss on both sides results from chiasmatic compression. This signature sign is pathognomonic, pointing to a pituitary tumor in adults or a craniopharyngioma in childhood. Cushing's chiasmatic syndrome consisted of bitemporal field defects, primary optic atrophy, and an unexpanded sella. This triad points to a suprasellar meningioma, rather than to the typical pituitary adenoma that enlarges the sella.[1]

Optic atrophy in one eye and papilledema in the other, the syndrome of Foster Kennedy,[2] points to a large frontal tumor, like a large meningioma. Central visual loss occurs on the side of the tumor and is due to primary optic atrophy from compression of the intracranial segment of the optic nerve. However, the patient tends to ignore the visual loss, inattention resulting from marked compression of the frontal lobe. Raised intracranial pressure causes choking of the opposite optic nerve head.[3] Peripheral visual loss, like looking through a knot-hole, occurs later, on the side of papilledema. An atrophic optic disc cannot swell,[4] but a swollen disc can atrophy. This secondary optic atrophy reduces the peripheral visual field.

While bitemporal hemianopia is a common pathognomonic sign of chiasmatic involvement, primary optic atrophy on one side and papilledema on the other is a rare pathognomonic combination. These signature signs indicate very specific locations of nerve compression in the nervous system. The actual causes of pinching of these nerves are immediately suspected by virtue of the relative incidence of tumors in these specific regions in different age groups. For example, pituitary tumors and meningiomas are rare in childhood. There is always a differential diagnosis with respect to actual cause. Gliomas occur within the optic nerves, chiasm, tracts, and the thalamus, generating specific visual field deficits. Gliomas are also common in the frontal lobes of adults, even more common than meningiomas, and gliomas can also produce the rare Foster Kennedy syndrome.[1,2]

A peripheral facial nerve palsy on the side of deafness, sometimes with trigeminal numbness on the same side, points unerringly to the cerebellopontine angle.[1] This combination of nerve signs is also pathognomonic, pointing to a tumor compressing the eighth, seventh, and fifth cranial nerves in the angle between the cerebellum and pons, and nowhere else. The actual cause is nearly always a benign tumor, usually an acoustic neuroma, sometimes a meningioma. Facial pain on the same side suggests a dermoid tumor. According to Webster's Dictionary, "pathognomonic" features are those "specially, distinctively, or decisively characteristic of a particular disease."[5] By this definition, bitemporal hemianopia is better called a signature sign of involvement of the optic chiasm, since very different diseases may be its cause. Pituitary tumors, craniopharyngiomas, meningiomas, optic gliomas, or tumors obstructing the third ventricle may be responsible for bitemporal hemianopia.[1] Aneurysm, trauma, and inflammation are occasional causes of this chiasmatic signature.[4] A signature sign points to a specific location in the nervous system, and nowhere else; it only suggests the "particular disease" responsible.

Harvey Cushing was correct to call bitemporal hemianopia the signature of the optic chiasm, and not a pathognomonic sign. Cushing doubtless discerned that "pathognomonic" is a troublesome word, too close in sound and spelling to "pathognomy" which Webster defines as "the study or recognition of emotions and passions through their outward signs or expressions."[5] No two words are so close, yet so distant, the one pointing to a specific organic disease, the other to unspecified emotional display. These words are pathological twins, one a giant, the other a dwarf.

Signature signs are not necessarily the most common signs of the causative lesion. Monocular blindness and a

---

CHARLES W. NEEDHAM, M.D., M.Sc., F.A.C.S., Staff neurosurgeon, Norwalk Hospital, Clinical faculty, Yale University School of Medicine.

crossed temporal field cut are as common as bitemporal hemianopia resulting from pituitary tumors that compress the optic chiasm. Meningiomas and gliomas involving the frontal lobe of the brain are usually diagnosed without optic atrophy on one side and papilledema on the other. As in the case of pituitary tumors, the diagnosis of frontal tumors is often made in the complete absence of optic nerve signs or visual field defects.[1] Seizures or the symptoms of raised intracranial pressure may bring frontal lobe tumors to diagnosis, while secondary amenorrhea and hypopituitarism point to tumors in the sella.[3]

The discovery of signature signs helps the busy physician to establish a clinical diagnosis before a computed tomographic scan (CT), MRI, or other confirmatory test is ordered. A signature sign points to a very specific locus in the nervous system; it does not really determine whether or not the actual cause is neoplastic, inflammatory, vascular, traumatic, etc. Signature signs commonly occur as a result of cranial nerve palsies due to lesions at the base of the skull.[6] For example, the orbital apex syndrome includes a variety of possible causes leading to the signature combination of oculomotor, trochlear, and abducens palsies and trigeminal pain on the side of visual loss and proptosis. A staring, paralyzed, and bulging eye, sometimes pulsatile, offers a striking appearance. The orbital apex is a very specific location, the point of passage of the neurovascular stem of the eye, at the junction of the anterior, middle, and pituitary fossae of the skull. Here, at the inner end of the sphenoid ridge, the ophthalmic artery and optic nerve pass through the optic foramen, and cranial nerves to the ocular muscles leave the cavernous sinus to pass in the superior orbital fissure, in the company of veins that drain the orbit. Various tumors, benign and malignant, including meningiomas, pituitary adenomas, metastatic cancers to the skull base, or retroorbital neoplasms or granulomas may be responsible. Large aneurysms of the carotid, aneurysms in the cavernous sinus and traumatic fistulas of the cavernous sinus, mucocele of the sphenoid sinus, sphenoiditis, etc., can produce the same picture of unilateral ophthalmoplegia, blindness, and pain.[1,7] Cranial nerve palsies are usually trustworthy in localizing causative lesions, with the exception of the abducens. Rarely, a curious syndrome of multiple bilateral cranial nerve palsies can result from large supratentorial tumors at a distance, due to a combination of translated intracranial mass effects.[8] However, the cranial nerves remain the treasure-house of signature signs and signature combinations that localize intracranial lesions of neurosurgical interest, including benign and malignant tumors, cysts, malformations, and aneurysms. Signature signs are the exact opposite of false-localizing signs. An abducens palsy cannot be trusted. The responsible mass may be far from the abducens nerve, even supratentorial, and not necessarily on the side of lateral rectus weakness. Or the abducens palsy can be diabetic, and totally unrelated to any intracranial mass. Indeed, the combination of primary optic atrophy on the one side and papilledema on the other is closely simulated by vascular disease involving the optic nerves.[2] While contraction of the peripheral visual field results from secondary optic atrophy, it may be simulated in patients with normal discs and psychogenic tunnel vision. Retinal "tilting of the optic discs" can simulate chiasmatic bitemporal visual field depression.[4]

A signature sign is an elite localizing sign: it points nowhere else. Objective neurologic signs confirm subjective symptoms, but even such objective physical signs require further validation, usually by CT or MRI, since such neurologic signs are sometimes simulated or falsely localizing. Babinski signs are lateralizing rather than localizing, implicating the brain or spinal cord. However, Babinski signs are sometimes bilateral, even when the upgoing toes result from unilateral intracranial or intraspinal causes. In contrast, cranial nerve palsies are usually both lateralizing and localizing, giving rise to exact signature signs pointing to an exclusive single locus within the head. Signature signs also result from compression of certain spinal nerve roots, often due to ruptured discs, spinal tumors, etc.

The "over-the-head" sign (see Fig. 1) is a new signature sign for a pinched lower cervical nerve root, usually the seventh, sometimes the sixth. The typical patient complains of pain at the base of the neck, radiating down the arm, sometimes as far as the hand and fingers. The peculiar "over-the-head" position of the involved arm is one in which the victim obtains a good measure of relief. The appearance is so odd, with one's symptomatic arm draped entirely over one's head, that the patient avoids the position in public and assumes it in private. Thus, the patient should be asked to show what position of the painful limb provides relief. The afflicted person then demonstrates the "over-the-head" sign, sometimes combined with forward flexion of the neck. The "over-the-head" position is the least uncomfortable position, whether the patient is erect or supine.

Most patients suffering a pinched cervical nerve root know exactly what aggravates their pain, the movements and postures to avoid, but not every patient is conscious of postures that help. One recent patient with neck and left arm pain, a right-handed art student, was quite unaware she was painting a canvas with her left arm draped over her head. A fellow art student in the same class quietly selected her as his subject, painting her with her right hand holding her brush and her left arm draped over her beret!

If the "over-the-head" sign occurs after neck injury, and routine x-ray films show no fracture, a ruptured cer-

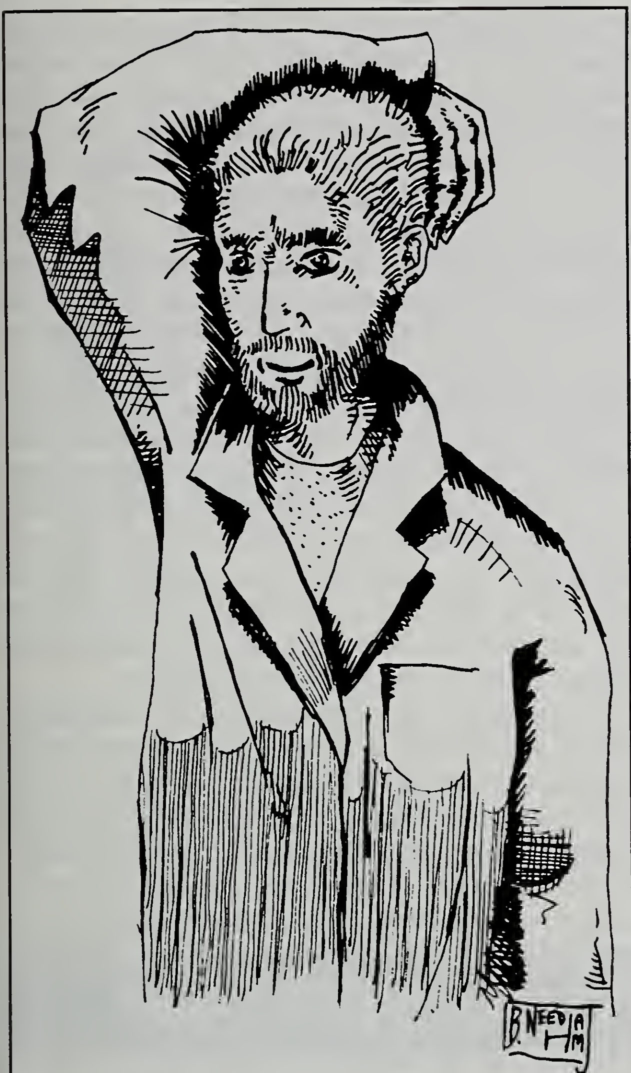


Figure 1.

vical disc becomes the most likely cause. The "over-the-head" sign differentiates these herniated disc cases from routine cervical muscular strains commonly seen in the emergency department and office. Ruptured cervical discs are most common between the sixth and seventh vertebra, pinching the C7 nerve root. Neck pain radiating to the arm, especially over the triceps muscle posteriorly, is common. Weakness of the triceps muscle and a reduced triceps reflex may be present. Pain of C7 root origin may radiate into the radial side of the forearm, with paresthesias in the index and middle fingers. Weakness of finger extensors, latissimus and pectoralis major, may be associated.[9] Under such circumstances, the "over-the-head" sign is truly pathognomonic of a ruptured C6–7 disc causing the C7 compressive radiculopathy. If the patient stops wincing and exhibits a sigh of relief as he places his painful arm over his head, we may conclude that the sign is emotionally pathognomic as well!

In contrast, the "over-the-shoulder" sign (see Fig. 2 and 3) is not a signature sign for any particular cervical nerve root. The patient is asked to indicate the painful area of the neck or shoulder. He places his good hand on the painful side, between the neck and the shoulder, his fingers over the trapezius. Pain of cervical nerve root origin is commonly referred along the medial edge of the scapula. However, a pinched C6 nerve root or C7 nerve root or C8 nerve root commonly refers pain from the neck downward along the medial scapula.[9] The ultimate destination of C6 root pain or paresthesia is the thumb (or thumb and index); C7 root pain or paresthesia is the index and middle finger; C8 root pain is the ring and little finger. Weakness of the deltoid points to the C5 root, the biceps the C6 root, the triceps the C7 root, and the intrinsic hand muscles the C8 root.[10] Weakness of

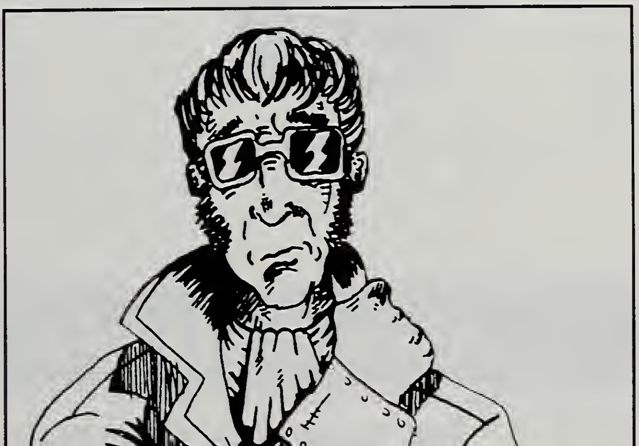


Figure 2.

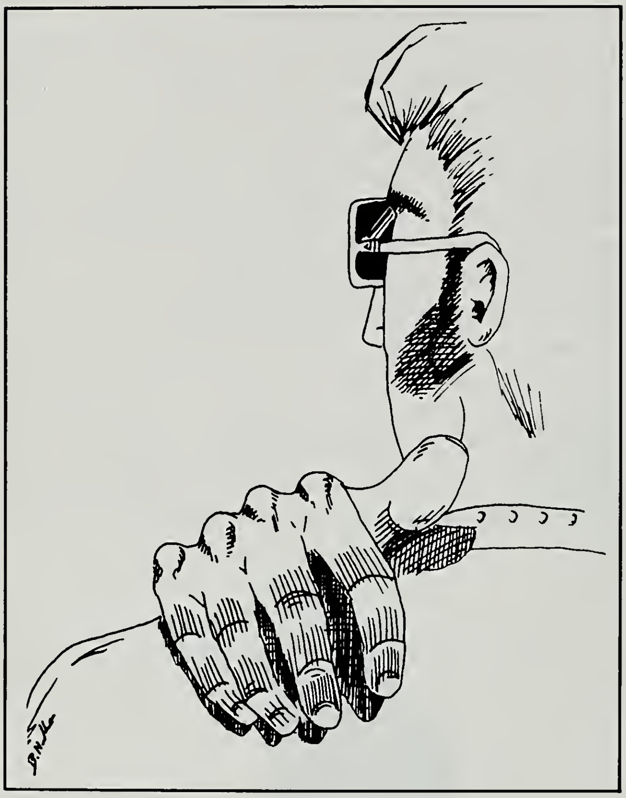


Figure 3.

wrist extension usually points to the C6 root, but is not pure C6.[9]

There are six cervical discs, seven cervical vertebra, and eight cervical nerves. There are no discs at the location of the C1 root between the occiput and atlas, or the C2 root between the atlas and axis. The C3 and C4 roots are rarely involved by disc ruptures.[9] C5 root pain extends to the deltoid, but the C4–5 disc ruptures that produce it are uncommon. Similarly, C8 root pain to the ulnar surface of the arm and ulnar two fingers resulting from a C7-T1 disc rupture is also uncommon.[9] C7 root pain, however, results from the most common C6–7 disc rupture, while C6 root pain is produced by the next most common C5–6 herniated disc. The posterior primary rami of the various cervical nerves supply the paravertebral muscles, so the medial scapular pain of the "over-the-shoulder" sign insufficiently distinguishes any particular cervical nerve root. The anterior primary rami of the various cervical spinal nerves (C5 through T1) supply the muscles of the upper limb through the brachial plexus.

The "over-the-shoulder" sign points to the cervical nerve roots but is otherwise unspecific. "Referred pain to the medial or upper scapula may be the only manifestation of segmental root involvement and typically reflects compression of the posterior primary ramus of the spinal nerve anywhere from C6 to T1."[9] This pain is referred along a dorsal line parallel to the spine, in the borderland between the shoulder joint and the joints of the spine. Cervical muscular strain (whiplash), muscle-contraction headache, and fibromyositis may simulate medial scapula nerve root pain. Chest pain that mimics angina occasionally results from lesions involving the C5 through C8 nerve roots.[9] Cervical nerve root pain radiating to the arm sometimes paradoxically spares the neck, but the cervical nerve root origin becomes clear by careful neurologic examination. For example, weakness of finger extensors and a reduced triceps reflex may be discovered. The opposite also occurs, with pain of cervical nerve root origin limited to the neck and medial scapula, sparing the arm. Such patients are commonly believed to suffer only cervical and thoracic muscular sprains, without radiculopathy. The unsuspected radiculopathy becomes obvious only after the neck pain proves intractable, and a CT scan or MRI reveals a ruptured cervical disc compressing the nerve root in the neural foramen.

Cervical spondylosis, osteoarthritis, and stenosis also produce similar clinical pictures and pinched cervical nerve roots.[10] A history of recent or remote trauma is not always given, even when a ruptured cervical disc is discovered to be the offending cause. Cervical trauma is common, and may worsen preexistent degenerative disc and joint disease. Various spinal tumors, especially neurofibromas, can produce the same picture of pinched nerves in the neck. Pain is the premier symptom of spinal nerve root origin, just as blindness implicates the optic nerve, and deafness the cochlear nerve. However, pain, like blindness and deafness, may arise from causes other than specific nerve involvement. The search for abnormal physical signs to document and localize the exact causes of such disabling symptoms is a stimulating exercise for the clinician.

The "over-the-head" and "over-the-shoulder" signs are quite unrelated to myelopathic signs, long-tract signs due to compromise of the spinal cord. Babinski signs, and other central nervous system—spinal cord signs, like gross motor paralysis of all limbs and urinary incontinence, point to the spinal cord, as distinct from particular cervical nerve roots. Nerve root signs implicate nerves outside the central neuraxis of the spinal cord, although their cause may lie within the bony spinal canal, next to the spinal cord. If the spinal cord is also compromised, then nerve root signs may be associated with myelopathic cord signs like the Babinski.

The spinal nerves, like the cranial nerves and autonomic nerves, comprise the peripheral nervous system. The "over-the-head" sign is a new signature sign of a pinched lower cervical nerve root, usually the seventh, sometimes the sixth. Easy compression of the extended fingers (C7) or the extended wrist (C6), and listening to the patient's exact description of the pain, makes the diagnosis in the office or at the bedside. Compression of cranial nerves and spinal nerves are our best sources of signature signs that point the way as surely as the needle of a compass.

REFERENCES

1. Needham CW: *Neurosurgical Syndromes of the Brain.* Springfield, Ill, Charles C. Thomas, 1973, pp246–9, 306–8.
2. Bender MB, Rudolph SH, and Stacy CB: The neurology of the visual and oculomoter systems. In: *Clinical Neurology.* Philadelphia: Harper and Row, 1985, vol 1, chap 12, p 9.
3. Needham, CW: *Neurosurgical Signs: Brain Management and Regional Diagnosis for Primary and Emergency Physicians.* Springfield, Ill, Charles C. Thomas, 1986, pp 17–20.
4. Mayo Clinic and Mayo Foundation: *Clinical Examinations in Neurology.* Philadelphia, Saunders, 1971, pp 75–83.
5. Webster's *Third New International Dictionary of the English Language,* Unabridged. Springfield, Mass, Merriam, 1966, p 1655.
6. Needham CW: Major cranial neuralgias and the surgical treatment of headache. In: *Medical Clinics of North America.* Philadelphia, Saunders, May 1978, p 62.
7. Roger J, Bille J, and Vigouroux RA: Multiple cranial nerve palsies. In: *Handbook of Clinical Neurology.* Amsterdam, North Holland, 1969, vol 2, chap 5, pp 86–8.
8. Needham CW, Bertrand G, and Myles ST: Multiple cranial nerve signs from supratentorial tumors. *J Neruosurgery* 1970;33:178–83.
9. Tsairis P and Jordan B: Neurological evaluation of cervical spine disorders. In: *Disorders of the Cervical Spine.* Baltimore, Williams and Wilkins, 1992, pp 11–22.
10. Lechtenberg R and Shah SK: Cervical myelopathy and root syndromes. In: *Disorders of the Cervical Spine.* Baltimore, Williams and Wilkins, 1992, pp 23–34.

Be Part of the Excitement!

HEALTHCARE EXPO '93

Sponsored By CHA The Connecticut Hospital Association and Affiliates

SAVE THE DATES: **APRIL 21 & 22, 1993**
HARTFORD CIVIC CENTER

- Over 285 exhibitors offering the most recent advances in products and services in the Healthcare Industry
- Travel giveaway
- **Special Physician Program** sponsored by The Connecticut State Medical Society
- Free Admission for all healthcare professionals

For More Information Call Robert Donnell Productions at 203-233-3654 • 800-243-9774

Because One Size Doesn't Fit All...

New Humulin 50/50 is the tailor-made answer to individual patient needs. A unique combination of equal amounts of Regular human insulin and NPH human insulin, it will be useful in situations in which a greater initial insulin response is desirable for greater glycemic control.

Like Humulin 70/30*, new Humulin 50/50 offers the convenience and accuracy of a premix. And it can be used in conjunction with an existing 70/30 regimen.

New Humulin® 50/50

50% human insulin isophane suspension
50% human insulin injection
(recombinant DNA origin)

The Newest Option in Insulin Therapy

WARNING: Any change of insulin should be made cautiously and only under medical supervision.

* Humulin® 70/30 (70% human insulin isophane suspension, 30% human insulin injection [recombinant DNA origin]).

Lilly

Global Excellence in Diabetes Care
Eli Lilly and Company
Indianapolis, Indiana
46285

HI-7911-B-249343 © 1992, ELI LILLY AND COMPANY

Interactive Effects of Basic Fibroblast Growth Factor and Heparin on Bone in 21-Day Fetal Rat Calvariae

MICHAEL N. KESSLER, B.A., LAWRENCE G. RAISZ, M.D., AND MARJA M. HURLEY, M.D.

ABSTRACT—We examined the interactive effects of heparin and basic fibroblast growth factor (bFGF) on collagen and DNA synthesis in 21-day fetal rat calvariae. In calvariae treated for 24h with heparin (25 μg/ml), a significant inhibition of methyl [^{3}H]thymidine (Tdr) incorporation into DNA and [^{3}H]proline labeling of collagenase-digestible protein (CDP) occurred compared to control. Treatment for 24h with bFGF (10^{-11} to 10^{-9}M) caused a stimulation of Tdr incorporation. With 96h treatment, bFGF (10^{-9}M) inhibited CDP labeling by 61%. Basic FGF in combination with heparin overcame the inhibitory effects of heparin on Tdr incorporation. The combination of bFGF plus heparin produced an even greater inhibition of CDP labeling than either effector alone. To assess the role of prostaglandin E_2 (PGE_2) in moderating the effects of bFGF, calvariae were treated with bFGF in the presence and absence of indomethacin (10^{-6}M), an inhibitor of PGE_2 production. Indomethacin did not alter the effects of bFGF on Tdr or CDP. We conclude that heparin and bFGF do interact to modulate collagen synthesis in bone via a PGE_2-independent mechanism.

Introduction

Basic fibroblast growth factor (bFGF) is an 18kD protein with a ubiquitous distribution.[1] The bFGF gene is located on human chromosome number eight and contains two introns and three exons. Two negative regulatory elements have been identified, one in the nontranscribed 5′ promoter region and one in the transcribed but not translated 3′ sequence of the promoter core. Three heparin binding domain residues have been discovered throughout the protein. It is known that exogenous bFGF is transported to the nucleus where it accumulates in the nucleolus. Once there, bFGF stimulates transcription of ribosomal genes during the G0-G1 transition phase of the cell cycle. In previous studies, bFGF has also been shown to induce mitogenesis in many different cell types including osteoblasts.[2,3]

Bone-derived bFGF is found associated with heparin sulfate in the extracellular matrix of tissues including bone. This has lead to the belief that bFGF may be involved in bone turnover.[4,5] During tissue injury, local release of heparin-like molecules and various proteases activate matrix-bound bFGF.[6,7] bFGF has significant homology with acidic fibroblast growth factor, and both factors stimulate replication of bone cells[8] and decrease osteoblast differentiation.[9]

Recent studies have shown that heparin alone inhibits collagen synthesis in bone,[10,11] while heparin combined with bFGF results in even greater inhibition.[12] It has been suggested that this inhibition may result from modulatory effects of heparin on bFGF activity.

Previous studies have suggested that some of the biological effects of bFGF may occur through the stimulation of prostaglandin E_2 (PGE_2) synthesis. In this study we examined the effects of bFGF on methyl[^{3}H]thymidine (Tdr) incorporation into DNA and [^{3}H]proline labeling of collagenase-digestible protein (CDP) in the presence and absence of indomethacin, a potent inhibitor of PGE_2 synthesis.

Methods

The method used for bone organ culture has been previously described.[13] Time-mated Sprague-Dawley rats were sacrificed on the 21st day of gestation. Dissection of the frontal and parietal bones of the fetal rats was then performed with division along the intervening suture line. Bones were cultured in 2 mL BGJ_b medium containing 1 mg/mL bovine serum albumin, 100 μg/mL ascorbic acid, 1 mM proline, and 0.01 mM thymidine.

MICHAEL N. KESSLER, B.A., Summer Researcher, The University of Connecticut Health Center, Farmington; LAWRENCE G. RAISZ, M.D., Professor and Head of Division; Endocrinology and Metabolism, The University of Connecticut Health Center, Farmington; MARJA M. HURLEY, M.D., Assistant Professor of Medicine, Division of Endocrinology and Metabolism, The University of Connecticut Health Center, Farmington.

This work is supported in part by NIH grant #AR-38933

Table 1

Effect of Heparin and bFGF on $[^3H]$Thymidine Incorporation into DNA (Tdr) in 24h Cultures

Group	*Tdr (dpm/μg dry weight)*
Control	73 ± 5
bFGF	
10^{-11}M	102 ± 11
10^{-10} M	132 ± 2
10^{-9} M	254 ± 25^a
Heparin 25 μg/mL	44 ± 5^a
+bFGF 10^{-11} M	75 ± 5
+bFGF 10^{-10} M	174 ± 15^a
+bFGF 10^{-9} M	301 ± 18^a

Values are the mean ± S.E. for 6-28 bones/group.
a = Significantly different from Control $P < 0.05$.

Heparin and bFGF, both in sterile water, were added at various concentrations.

DNA Synthesis.—To assess DNA synthesis, calvariae were treated with effectors for 24h and were incubated with methyl $[^3H]$thymidine (Tdr 5 μCi/mL, 76.7 Ci/mmol) during the last 2h of the culture period. At the end of the incubation, the bones were rinsed in cold BGJ medium, blotted on filter paper, and extracted with 0.5 mL of 5% trichloroacetic acid (TCA). Acid-extractable $[^3H]$Tdr was determined by measuring the radioactivity in the TCA washes. Each calvaria was extracted with acetone and ether, dried and weighed, and hydrated in 1 mL of 0.5M acetic acid for at least 1h. Calvariae were homogenized, and 100 μL aliquots were counted in liquid scintillation fluid. Cold TCA insoluble counts were taken as a measure of Tdr incorporation into DNA.

Collagen Synthesis.—Calvariae were transferred to fresh medium daily and radiolabeled with $[^3H]$proline (5 μCi/mL) for the final 2h of the culture period. Each calvaria was washed in 1 mL 0.5M acetic acid. To quantitate the incorporation of $[^3H]$proline into collagenase-digestible protein (CDP) and noncollagen protein (NCP), homogenates were digested with purified bacterial collagenase.[14] The percent collagen synthesis (PCS) was corrected for the relative abundance of proline in collagen and noncollagen proteins.[15]

Statistics.—All data were analyzed with a one-way analysis of variance. Scheffe's LSD method was used to make pairwise comparisons.

Results

The effects of various concentrations of bFGF were examined in the presence and absence of 25 μg/mL of heparin to determine the effect on DNA synthesis (Table 1). At 24h bFGF (10^{-11} to 10^{-9}M) increased Tdr incorporation from 139 to 347% in a dose dependent manner compared to control. Heparin alone significantly inhibited Tdr. The addition of bFGF (10^{-11} to 10^{-9}M) was able to overcome the inhibitory effect of heparin on Tdr incorporation. Additionally, the heparin and bFGF (10^{-11} to 10^{-9}M) combination resulted in a greater, though not statistically significant, stimulation of Tdr incorporation than with bFGF (10^{-10} to 10^{-9}M) alone.

The rate of collagen synthesis was assessed by measuring the rate of $[^3H]$proline incorporation into CDP (Table 2). At 96h, bFGF (10^{-9}M) inhibited CDP labeling while bFGF (10^{-10} and 10^{-11}M) had no effect. Heparin alone inhibited CDP labeling to 52% of the control value. When combined with bFGF (10^{-11} to 10^{-9}M), heparin caused even greater inhibitory effects by bFGF. NCP remained unaffected in all groups.

We determined the effects of bFGF on collagen synthesis in the presence and absence of indomethacin (Table 3). Indomethacin alone had no effect on Tdr or CDP. bFGF at 10^{-9}M significantly stimulated Tdr by 247% and inhibited the CDP and PCS from the control value by 33% and 50% respectively. The addition of indomethacin (10^{-6}M) did not alter these effects.

Discussion

The inability of heparin to block the stimulation of Tdr by bFGF indicates that heparin's inhibitory effects are not due to the inactivation of endogenous bFGF. We have demonstrated that very low concentrations of bFGF inhibit CDP labeling when heparin is present. Furthermore, we have shown that while heparin alone inhibits Tdr incorporation, the combination of low concentra-

Table 2

Effect of Heparin and bFGF on $[^3H]$Proline Incorporation into Collagenase-Digestible Protein (CDP), Noncollagen Protein (NCP), and Percent Collagen Synthesis (PCS) in 96h Cultures

Group	*CDP (dpm/μg dry weight)*	*NCP*	*PCS (%)*
Control	23 ± 2	25 ± 2	14.1 ± 0.6
bFGF			
10^{-11} M	23 ± 2	26 ± 2	13.2 ± 0.8
10^{-10} M	22 ± 2	31 ± 2	11.3 ± 0.8^a
10^{-9} M	14 ± 2^a	30 ± 2	8.7 ± 1.4^a
Heparin 25 μg/mL	12 ± 1^a	23 ± 1	9.2 ± 0.4^a
+bFGF 10^{-11} M	8 ± 2^a	25 ± 2	5.4 ± 1.0^a
+bFGF 10^{-10} M	10 ± 2^a	28 ± 3	6.2 ± 0.3^a
+bFGF 10^{-9} M	6 ± 1^a	28 ± 2	3.6 ± 0.3^{a,b}

Values are the mean ± S.E. for 6-28 bones/group.
a = Significantly different from Control $P < 0.05$.
b = Significantly different from Heparin $P < 0.02$.

Table 3

Effect of bFGF (10^{-9} M) and Indomethacin (10^{-6} M) (Indo) on [^{3}H]Thymidine Incorporation into DNA (Tdr) in 24h Cultures and [^{3}H]Proline Incorporation into Collagenase-Digestible Protein (CDP), Noncollagen Protein (NCP), and Percent Collagen Synthesis (PCS) in 96h Cultures

Group	*Tdr*	*CDP (dpm/μg dry weight)*	*NCP*	*PCS (%)*
Control	73 ± 5	18 ± 1	25 ± 1	11.7 ± 0.5
+bFGF	254 ± 25[a]	12 ± 1[a]	34 ± 2[a]	5.9 ± 0.2[a]
Indo	74 ± 1	15 ± 1	22 ± 2	11.5 ± 0.6
+bFGF	285 ± 17[a]	9 ± 1[a,b]	30 ± 2	5.4 ± 0.3[a,b]

Values are the mean ± S.E. for 3-28 bones/group.
[a] = Significantly different from control $P < 0.05$.
[b] = Significantly different from Indo alone $P < 0.05$.

tions of bFGF with heparin eliminates this inhibition of Tdr. It is possible that heparin's prolongation of the half-life of bFGF is at least partially able to explain these results.[16] Recent studies have shown that bFGF is biologically active only when bound to high affinity cell surface receptors.[17,18] In order for this activating binding to occur, bFGF must initially bind to low affinity membrane-associated heparan sulfate proteoglycans. Exogenous heparin may substitute for these cell surface proteoglycans.[19]

Additional studies have demonstrated that one can alter the affinity of bFGF for heparin resulting in altered biological responses through site-directed mutagenesis of the heparin binding regions of the bFGF gene.[20] Enzymatic degradation or inhibition of sulfation of heparin also effects binding of bFGF to its receptor. As a result the mitogenic activity of bFGF in fibroblasts is absent.[21] These studies all suggest that bFGF's effects on tissues is directly associated with heparin or heparan sulfate.

The use of indomethacin has ruled out a significant role of PGE_2 as a mediator of bFGF's effects since blocking PGE_2 production did not alter the effects of bFGF on DNA or collagen synthesis.

REFERENCES

1. Gospodarowicz D: Basic science and pathology of fibroblast growth factor, chemical structure and biological function. *Clin Orthop Rel Res* 1990;257:231–48.
2. Goustin AS, Leof EB, Shipley GD, et al: Growth factors and cancer. *Cancer Res* 1986;46:1015–29.
3. Sporn MB, Todara G: Autocrine secretion and malignant transformation of cells. *N Enql J Med* 1980;303:878–80.
4. Hauschka PV, Maurakos AE, Jafrati MD, et al: Growth factors in bone matrix: Isolation of multiple types by affinity chromatography on heparin sepharose. *J Biol Chem* 1986;261:12665–74.
5. Globus R, Plouet J, Gospodarowicz D: Cultured bovine bone cells synthesize fibroblast growth factor and store it in their extracellular matrix. *Endocrinology* 1989;124:1539–47.
6. Bashkin P, Doctrow S, Klagsburn M, et al: Basic fibroblast growth factor binds to subendothelical extracellular matrix and is released by heparitinase and heparin like molecules. *Biochemistry* 1989;28:1737–43.
7. Saksela O, Rifkin D: Release of basic fibroblast growth factor-heparin sulfate complexes from endothelial cells by plasminogen activater-mediated protedytic activity. *J Cell Biology* 1990;110:767–75.
8. Globus RK, Patterson-Buckendahl P, Gospodarowicz D: Regulation of bovine bone cell proliferation by fibroblast growth factor and transforming growth factor B. *Endocrinology* 1988;123:98–105.
9. Rodan SB, Wesolowski G, Thomas K, et al: Growth stimulation of rat calvarial osteoblastic cells by acidic fibroblast growth factor. *Endocrinology* 1987;121:1917–23.
10. Hurley MM, Gronowicz G, Kream BE, et al: Effect of heparin on bone formation in fetal rat calvaria. *Calcif Tissue Int* 1990;46:183–8.
11. Hurley MM, Kream BE, Raisz LG: Structural determinants of the capacity of heparin to inhibit collagen synthesis in 21 day fetal rat calvaria. *J Bone Min Res* 1990;11:1127–33.
12. Canalis E, Centrella M, McCarthy T: Effect of basic fibroblast growth factor on bone formation in vitro. *J Clin Invest* 1988;81:1572–77.
13. Kream BE, Smith MD, Canalis E, et al: Characterization of the effect of insulin on collagen synthesis in fetal rat bone. *Endocrinology* 1985;116:296–302.
14. Peterkofsky B: The effect of ascorbic acid on collagen polypeptide synthesis and proline hydroxylation during the growth of cultured fibroblasts. *Arch Biochem Biophys* 1972;152:318–28.
15. Diegelman RF, Peterkofsky B: Collagen biosynthesis during connective tissue development in chick embryo. *Dev Biol* 1972;28:444–53.
16. Gospodarowicz D, Cheng J: Heparin protects basic and acidic FGF from inactivation. *J Cell Physiol* 1986;128:475–84.
17. Ullrich A, Schlessinger J: Signal transduction by receptors with tyrosine kinase activity. *Cell* 1990;61:203–12.
18. Sakaguchi K, Yanagishita M, Takeuchi Y, et al: Identification of heparan sulfate proteoglycan as a high affinity receptor for acidic fibroblast growth factor (aFGF) in a parathyroid cell line. *J Biol Chem* 1991;226:7270–8.
19. Yayon A, Klagsburn M, Esko JD, et al: Cell surface, heparin-like molecules are also required for binding of basic fibroblast growth factor to its high affinity receptor. *Cell* 1991;64:841–8.
20. Heath WF, Cantrell AS, Mayne NG, et al: Mutations in the heparin-binding domains of human basic fibroblast growth factor alter its biological activity. *Biochem* 1991;30:5609–15.
21. Rapraeger AC, Krufka A, Olwin BB: Requirement of heparan sulfate for bFGF mediated fibroblast growth and myoblast differentiation. *Science* 1991;252:1705–8.

For certain categories of taxpayers, we are still accepting contributions that will be deductible for 1992.

ENDORSED BY THE MEDICAL SOCIETY OF NEW JERSEY

**V.E.B.A. PLAN*

FEATURES

***TAX
DEDUCTIBLE
CONTRIBUTIONS
TO AN
I.R.S. APPROVED
TRUST***

EVEN IF ...

- **YOUR PENSION PLAN IS OVER FUNDED**
- **YOUR PENSION PLAN IS MAXIMUM FUNDED**
- **YOU HAVE NO PENSION PLAN**

PLEASE SEND ME A copy of your informational pamphlet on the * V.E.B.A. program.
I understand there is no obligation.

NAME______________________________

ADDRESS______________________________

DATE OF BIRTH________________ TELEPHONE # (____)________________

Mail to:

THE KIRWAN COMPANIES
402 MIDDLETOWN BLVD., SUITE 202
LANGHORNE, PA. 19047

(800) 283-7666........................ (215) 750-7616........................ FAX (215) 750-7791

*VOLUNTARY EMPLOYEE BENEFITS ASSOCIATION

Computed Tomography in Pulmonary Tuberous Sclerosis

VINCENT L. PEREZ, M.D. AND PETER M. SHIMKIN, M.D.

ABSTRACT—Pulmonary disease is a rare manifestation of tuberous sclerosis. Respiratory symptoms often progress to severe pulmonary insufficiency and death may ensue within a few years of diagnosis.[1] We describe a case where a computerized tomographic scan aided early diagnosis in a patient with spontaneous pneumothorax and subtle, nonspecific changes on a plain x-ray film.

Case Report

A 34-year-old women with known tuberous sclerosis presented with sudden shortness of breath and right sided chest pain. She had no history of trauma or previous pulmonary disease. A chest roentgenogram (Fig. 1) showed a right sided tension pneumothorax with a few thin walled left perihilar cysts. The periphery of the left lung appeared normal. Her pneumothorax persisted despite closed thoracostomy with negative pressure through a chest tube. Thoracic computed tomography showed both lungs filled with cysts of variable size and wall thickness (Fig. 2) establishing the diagnosis of pulmonary tuberous sclerosis. Open thoracotomy for pleurodesis revealed innumerable minute pulmonary blebs. An incidental wedge biopsy disclosed pulmonary blebs and abnormal proliferation of perivascular smooth muscle. The pneumothorax resolved and the patient was asymptomatic at discharge.

Discussion

Tuberous sclerosis is an uncommon heredofamilial disease. Although classically characterized by the clinical triad of mental retardation, seizure disorders, and skin lesions known as adenoma sebaceum, the triad is complete only in approximately 29% of patients.[1] Hamartomatous lesions are always present in the midbrain, especially in the subependymal region where they are known as tubers. Hamartomas in other organs include renal angiomyolipomas and cardiac rhabdomyomas.[1]

Clinical pulmonary involvement is rare, occurring in 0.1–1.0% of patients.[2] The histologic findings are thin walled cysts which vary in size from a few millimeters to centimeters, and perivascular smooth muscle proliferation between the cysts. The pulmonary vessels and lymphatics are normal. In florid cases the chest roentgenogram will show prominent interstitial markings, cysts, and/or a honeycomb pattern. In more subtle cases com-

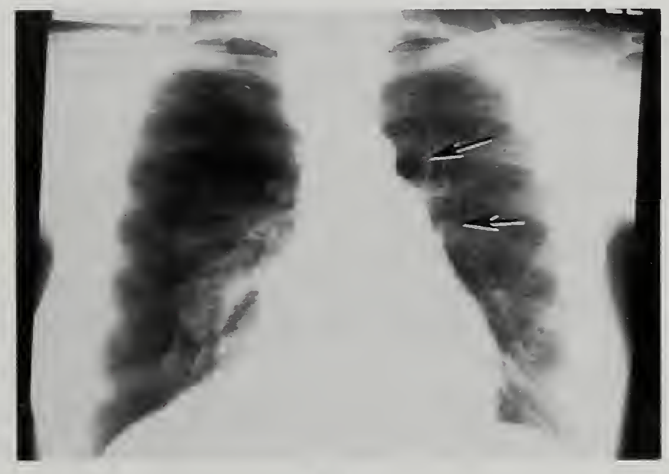


Figure 1

Postero-anterior chest roentgenography reveals a large right pneumothorax and left perihilar parenchymal cysts (arrows).

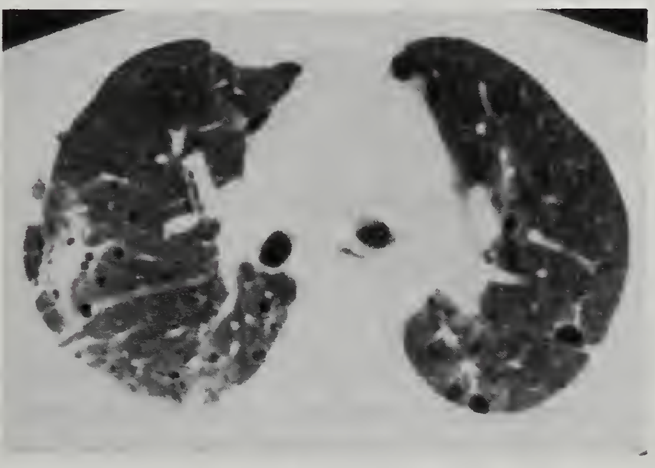


Figure 2

High resolution computed axial tomography of the chest obtained after pleurodesis reveals numerous bilateral pulmonary cysts of varying size and wall thickness. A right wedge resection defect and residual pneumothorax are also present.

VINCENT L. PEREZ, M.D., radiology resident, Bridgeport Hospital; PETER M. SHIMKIN, M.D., attending radiologist and director of neuroradiology at Bridgeport Hospital.

puted tomography will easily demonstrate characteristic cysts of varying size and wall thickness. This, combined with clinical history and physical findings, establishes the diagnosis and biopsy is unnecessary.[3]

REFERENCES

1. Lie JT: *Tuberous Sclerosis, Second Edition.* Ed. M. Gomez, New York, Raven Press, 1988, pp 150–60.
2. Slingerland JN, Grossman RF, Chamberlain D, et al: Pulmonary manifestations of tuberous sclerosis in first degree relatives. *Thorax* 1989;44:212–4.
3. Lenoir S, Grenier P, Brauner MW, et al: Pulmonary lymphangiomatosis and tuberous sclerosis: Comparison of radiographic and thin section CT findings. *Radiology* 1990; 175:329–34.
4. Fraser R, Pare JA, Pare PD, et al: "Tuberous Sclerosis," In: *Diagnosis of Diseases of the Chest, Third Edition.* Philadelphia, W.B. Saunders Company, 1991, pp 2677–9.

THE AMERICAN SUICIDE FOUNDATION
and
McLean Hospital Department of Postgraduate and Continuing Education
present

Suicide In Medical and Surgical Practice: Prevention and Management

John Hancock Building • Quincy Suite • 180 Berkley Street • Boston, Massachusetts

Topics

Suicide: A Persepective
Recognition of the Suicidal Patient
Psychopharmacology and Suicide
Physician Assisted Suicide: Danger of Legalization
Suicidal Patients: Problems in Risk Management

Faculty

Thomas G. Gutheil, M.D.
Herbert Hendin, M.D.
John T. Maltsberger, M. D.
George E. Murphy, M.D.
Alex Nicholas Sabo, M.D.

McLean Hospital designates this continuing medical activity for 5.5 credit hours and is accredited by The Accreditation Council for Continuing Medical Education.

Registration is $50.00 per person. For more information call Nancy or Shirley at 617-965-2664.

Psychopharmacology of Nicotine: Implications for Pharmacotherapy in Psychiatry

ALEX A. CARDONI, M.S. PHARM.

It is better to be without a wife for a bit than without tobacco for an hour. Estonian Proverb

WHILE you may not agree with this aphorism, it does emphasize the power of tobacco over humans. And indeed, in 1992, the power of tobacco continues to be manifested worldwide. In 1990, 50 million American adults still smoked with men (32%) smoking more than women (27%).[1] Despite these numbers, they represent a substantial decrease (50%) in the numbers of smokers since 1964, the year the surgeon general, Dr. Luther Terry, issued the first report on the health effects of smoking. Subsequent reports summarized research findings that showed that smoking causes approximately 430,000 deaths per year. Forty-six percent are related to cardiovascular disease, 33% to cancers, 19% to respiratory diseases, and 2% to other causes.[2] We know that a 35-year-old one to two pack-per-day (PPD) smoker will have life expectancy shortened by seven years with continued smoking. Six of these years can be regained by smoking cessation.[3] But it is difficult to stop smoking, as we are well aware. Of the 17 million adults who attempt to quit each year, only 8% are successful.[1]

A review of the pharmacology and pharmacokinetics of nicotine, the addicting substance in tobacco, will contribute to a better understanding of why it is so difficult for people to stop smoking. Furthermore, such a review will clarify why successful pharmacotherapy of the smoking psychiatric patient may be difficult or unattainable because of pharmacokinetic and pharmacodynamic interference by nicotine. Finally, the advent of transdermal nicotine has increased the chances of successful nicotine withdrawal so that the goal of an American smoke-free society by the year 2000, as targeted by former Surgeon General C. Everett Koop, is within reach.

What's In Tobacco Smoke?

The tobacco leaf contains thousands of chemicals that are carried in smoke when the tobacco is burned. The usual cigarette "dosage form" contains 15–40 mg of particulate matter ("tar"), 10–25 mg carbon monoxide, 1–2 mg nicotine, and 0.5–1.0 mg acetaldehyde. The polycyclic hydrocarbons anthracene and benzpyrene, N-nitrosamines, and polonium 210 are the known carcinogens in tobacco smoke. The hydrocarbons are responsible for the induction effects on the P-449 and P-450 hepatic microsomal enzyme systems. Nicotine has no significant enzyme induction activity. Induction results in increased activity of these systems which are responsible for oxidative metabolism of both endogenous chemicals manufactured by the body and xenobiotics ingested in the form of drugs and other chemicals. This mechanism is believed to be operative in the weight loss or lack of weight gain commonly seen in smokers and the weight gain that usually accompanies smoking cessation.

These effects are not obviated by the use of smokeless tobacco. Chewing tobacco and snuff contain significant amounts of nicotine alkaloids (0.5–5%), alkanes (0.1–0.4%), terpenes (0.1–3%), and polyphenols (0.5–4.5%). They also contain N-nitrosamines, polycyclic hydrocarbons, and polonium 210. Significant absorption occurs through the oral mucosa resulting in potent systemic effects.

Smoking—Drug Interactions

Enzyme induction from tobacco use accelerates the metabolism of many drugs. This results in more rapid clearance, lower blood levels, diminished response, and potential therapeutic failure. The induction effects occur over a period of several weeks and the magnitude of the effect depends on the "dose" of inducing agent(s) delivered to the enzyme. Virtually all smoking-drug interaction studies have included subjects that smoke at least one PPD. Resolution of this effect also occurs over a period of weeks after cessation of tobacco use. Thus, dosage of medication(s) affected will have to be adjusted downward to avoid side effects and possible toxicity. The

---

ALEX A. CARDONI, M.S. PHARM., Associate Professor of clinical pharmacy, the University of Connecticut, Storrs; Assistant Clinical Professor of psychiatry, the University of Connecticut Health Center, Farmington; Clinical Psychoparmacology Consultant, department of psychiatry, Hartford Hospital, Hartford.

Previously published in the Hartford Psychiatric Letter, a publication of the Department of Psychiatry at Hartford Hospital.

following is a list of medications that will show diminished response in smokers:

- acetaminophen
- antidepressants
- phenothiazines
- beta blockers
- estrogens
- glutethimide
- haloperidol
- heparin
- insulin
- oral contraceptives
- pentazocine
- phenylbutazone
- propoxyphene
- theophylline

Nicotine Pharmacology

Nicotine is an excitatory chemical which affects cholinergic, adrenergic, and dopaminergic systems in the body. Centrally, nicotine facilitates cholinergic transmission by binding to pre- and postsynaptic "nicotinic" receptors. Presynaptic binding promotes acetylcholine release from storage granules while postsynaptic binding produces a direct cholinergic agonist effect. Nicotine produces stimulation, increased vigilance and concentration, and improved memory. The hedonic experience associated with tobacco use is thought to be caused by nicotine-mediated increased limbic dopamine activity and increased hypothalamic luteinizing hormone-releasing hormone secretion. Peripherally, the effects of nicotine are primarily cardiovascular. Increased heart rate mediated by increased pressor amine release via adrenal, peripheral sympathetic, and peripheral ganglion stimulation is the predominant effect.

Nicotine Pharmacokinetics

Nicotine most often enters the body via inhalation which is the most rapid route of administration. Inhaled nicotine passes directly into arterial blood and is carried to central sites of action within five to seven seconds. The amount of nicotine delivered to the alveolar capillary membrane depends on the amount inhaled, the depth of inhalation, the duration of inhalation, the pH of the smoke, and individual absorption characteristics of the smoker. Thus, the amount of nicotine ingested can be controlled by the smoker and indeed this is done to maintain a "comfort zone" nicotine blood level. Significant nicotine levels can be achieved with chewing tobacco and snuff, although the time to peak levels is longer. However, levels remain high as long as the tobacco is kept in the mouth, creating a kind of sustained-release dosage form. Indeed, the "bioavailability" of a "wad" of chewing tobacco or a "plug" of snuff greatly exceeds that for one cigarette. In this respect, these products are not safer, as the manufacturers would have the public believe.

Nicotine is rapidly metabolized in the liver with 70% of a dose converted to cotinine. All known metabolites are excreted in the urine as weak bases and therefore elimination depends on urine pH. In an acidic urine, the clearance of nicotine can be increased by 12-fold. Smokers probably unwittingly compensate for increased or decreased excretion of nicotine by adjusting their smoking to maintain the "comfort zone" blood level.

Nicotine Dependence and Withdrawal

The central effects of nicotine in producing a sense of pleasure, stimulation, and reward lead to a pattern of compulsive tobacco use. The addicting power of nicotine is revealed in examination of relapse rates over time for persons addicted to heroin, alcohol, and nicotine. Twelve-month relapse rates for heroin and nicotine are virtually superimposable. Both are worse than for alcohol users. These findings are not surprising when one considers what happens when people inhale tobacco smoke. The average standard filter cigarette delivers about 10–12 doses of nicotine (one "drag" = 1 dose). Each time a smoker takes a drag, 0.1–0.2 mg nicotine is delivered to the alveolar capillary membrane where most of it is absorbed and delivered to brain target neurons within five to seven seconds. If a person smokes one pack of cigarettes per day, between 200–240 "hits" of nicotine will be delivered to target neurons each day. If the smoker smokes two packs per day, this doubles to 400–480 EACH DAY. In sharp contrast, the average heroin user can usually get by with one to two "fixes" per day. This repetitive "bombing" of the neurotransmitter systems that results in stimulation, reward, and pleasure contributes significantly to the high relapse rates in tobacco cessation.

Nicotine withdrawal is characterized by both subjective and objective, or physical, effects. These include the following:

Subjective
- tobacco craving
- increased tension
- restlessness
- irritability
- aggressiveness
- depression
  - decreased concentration
  - increased hunger

Physical
- EEG changes
  - decrease in heart rate & BP
  - increased peripheral circulation
- sleep disturbance
  - increased weight
  - decreased performance on:
    - vigilance tasks
    - simulated driving
    - selective attention tasks
    - memory tasks

Withdrawal occurs within minutes to hours after the last cigarette, depending on the individual smoker. Indeed, the "need to have a cigarette" represents the need to use nicotine to prevent progression of the nicotine withdrawal syndrome. Smokers "feel better" after a smoke because they have restored their nicotine levels to the "comfort zone." The first cigarettes in the morning represent the need to restore the nicotine level to the comfort zone after no nicotine ingestion during sleep. With a half-life of elimination of two hours, nicotine levels have fallen close to zero overnight.

Treatment of Nicotine Withdrawal

Research data indicate that 60% of smokers who quit without the aid of a formal treatment program relapse within 30 days. This increases to 70% at 60 days and 80% at 90 days. A small percentage (13%) are able to stop on their own and remain abstinent for up to one year.[5]

The advent of nicotine dosage forms to facilitate nicotine withdrawal has prompted development of more formal treatment programs to assist smokers in tobacco cessation. Nicotine polacrilex (nicotine gum) was marketed in 1987 to help smokers separate the smoking behavior from the nicotine addiction. It has been helpful to some but suffers from several drawbacks. First, the rate of nicotine delivery is variable, depending on how the user chews the gum. It is supposed to be chewed slowly and "parked" in the buccal cavity over a twenty minute period to avoid unpleasant side effects of nicotine. Second, users can use the gum like a cigarette, chewing whenever they have a need to ingest nicotine.

Transdermal nicotine (nicotine "patch") is an improvement over the gum. The user places the patch on the skin for a sustained release of nicotine over a 16–24 hour period. The patches come in different strengths (21 mg, 14 mg, 7 mg) and the dosage of nicotine can be matched to the level of smoking. Transdermal nicotine also ensures that nighttime drops in the nicotine blood level will not occur. The patch has been compared to placebo, both with behavioral support, and abstinence rates are significantly higher with the patch (54% patch *vs* 30% placebo 7 weeks and 43% *vs* 30% 10 weeks). Craving scores were significantly higher for placebo subjects compared to transdermal nicotine subjects at each week of the seven week study.[6]

It is important to emphasize that the patch alone will not ensure successful cessation. It is not the "magic bullet." It is a useful tool that when combined with a program of behavioral support can significantly increase the chances of successful smoking cessation.

Implications for the Psychiatric Patient

Psychiatric patients as a group have the largest percentage of smokers (80–90%) when compared to other illness categories. Why is this? Are patients treating their illness with nicotine? Is the illness superimposed over a preexisting addiction? There is no doubt that the powerful CNS effects of nicotine can stimulate, provide a feeling of well-being, increase alertness, and sharpen concentration. But they can also disturb the sleep cycle, worsen psychotic symptoms, and exacerbate hypomanic behavior. There are good data to suggest an association between cigarette smoking and tardive dyskinesia, based on nicotine's effect in facilitating dopamine synthesis and release in animal basal ganglia.[7]

Psychiatric health professionals are in a dilemma over tobacco use by their patients. On one hand we should not be enabling behavior that is inconsistent with health; on the other, we don't want to take away an activity that is enjoyable to some patients. Knowledge of nicotine psychopharmacology can help us to (1) understand patient smoking behaviors as related to the course of their illness and response to psychotropic medication; and (2) understand aspects of their illness that may be related to CNS nicotine effects. Likewise, increased patient knowledge of nicotine effects on the brain can improve the acceptance of smoking as a nicotine dependency which is treatable.

Summary

Cigarette smoking is inconsistent with good health. In psychiatric patients it may also impede recovery through direct CNS effects of nicotine and indirect kinetic effects of enzyme-inducing agents. Psychiatric health professionals should promote smoking cessation with patient education and use of transdermal nicotine coupled with behavioral support.

REFERENCES

1. Fiore MC, et al: Methods used to quit smoking in the Unites States. *JAMA* 1990;263:2760–5.
2. Center for Disease Control: Smoking attributable mortality and years of potential life lost—United States. HHS Publication No. (CDC) 91-8017, Atlanta, GA, 1988.
3. U.S. Centers for Disease Control 1987, 1988, 1990 data; National Center for Health Statistics 1988 data; National Safety Council 1989 data.
4. U.S. Department of Health and Human Services: The health consequences of smoking nicotine addiction. A report the Surgeon General. DHHS Publication No. (CDC) 88-8406.
5. Sachs DPL: "Advances in Smoking Cessation Treatment," In: *Current Pulmonology*. Ed. Simmons, Year Book Medical Publishers, 1991, pp. 139–98.
6. Data on file, Ciba-Geigy Corporation, Summit, New Jersey, 1992.
7. Yassa R, et al: Nicotine exposure and tardive dyskinesia. *Biol Psychiat* 1987;22:67–72.

COMMUNITY HEALTH CENTERS

The Ultimate Health Career!

CONNECTICUT COMMUNITY HEALTH CENTERS have immediate employment opportunities for Board Eligible/ Board Certified Physicians in all primary care specialties.

POTENTIAL BENEFITS INCLUDE:

- School Loan Repayment
- Competitive Income and Benefits
- Multi-Disciplinary Team Care
- Focus On Patient Care
- Community and Public Service
- Full Administrative Support System

Please Contact: Lorraine Ahearn, Connecticut Primary Care Association, 30 Arbor Street, North Bldg., Hartford, CT 06106, (203) 232-3319 or Fax your C.V. (203) 236-0618.

The Choice Is Ours

PATRICK J. CAROLAN, M.D. AND VINCENT J. CATRINI, M.D.

ABSTRACT—Unlike their colleagues in all the other states, CSMS physicians are uniquely well positioned for the health care "reform" measures that may be coming down the pike from Washington and/or Hartford, thanks to their proactive organization of the state's only statewide IPA and HMO, CSMS-IPA and M.D. Health Plan. Now in the sixth year of operation, the former has grown to some 3,500 physicians, and the latter to some 75,000 enrollees.

Fortunately, the greater than budgeted utilization experienced earlier in 1992 has begun to resolve, and the withhold will be returned to 20% in January 1993. But to assure future security, the organizations have determined that such swings in utilization must be made less likely and less severe by individual physicians being informed at the local level where they stand with respect to preestablished clinical norms and to the performance of their peers.

Introduction

IN the not too distant future, it will become unrealistic to expect that physicians can maintain their current living standards while practicing business-as-usual medicine. Managed care is having a compelling effect on the practice of medicine, and new federal antitrust regulations are forcing physicians to disengage from related business activities that heretofore were permissible alternative income supplements. Traditional referral patterns are changing as doctors are required to adapt to health service delivery systems and provider networks designed by outsiders. Because these designs seek to control medical cost with little concern for patient care continuity, by definition certain physicians designated as "preferred" will win, while others are excluded, and therefore precluded from treating these patients. Eventually, the delivery network and referral patterns will be defined by non-MDs. This fragmentation will erode the influence that physicians have over the delivery of health care and its current emphasis on patients' needs.

Unlike any other state, physicians in Connecticut have a choice. M.D. Health Plan by definition has a single agenda, and that is to guarantee that physicians have a say in the way managed care affects their practice. No other HMO or insurance company can make that guarantee because none is exclusively owned by physicians. However, as HMO owners, CSMS physicians are in direct, open competition with these larger, older, and better capitalized HMOs and insurance companies having less appreciation of clinical issues, little concern for the patient/physician relationship, and few scruples about cutting corners on coverage and compensation. In spite of this, M.D. Health Plan continues to compete and succeed. But physicians must understand the price of this competition.

The medical cost structure of the premium, to a large extent, is a function of the design of the delivery system, the practice patterns of its providers, and the cost of each component. The medical cost structure represents 85-90% of the premium.

In a free market society, competition is encouraged and buyers (employers) will compare price (premiums) and select that plan with the lowest premium and the highest quality. Because quality in health care is difficult to define and measure, consumers resort to other factors such as; physician selection, health benefits, plan administration.

Comparing M.D. Health Plan to other HMOs, the employer may conclude that:

- plan administration is similar among HMOs;
- the health benefits are not remarkably different (this is mandated by law);
- most importantly, the physician network is not remarkably different although M.D. Health Plan is more geographically dispersed.

Without perceived differences in these intangible areas, price (premium) becomes the only determinant.

HMOs vary their tactics to achieve their cost-containment objectives. Many may be in conflict with physicians' practice patterns. M.D. Health Plan deliberately avoids some of the fundamental constraints, such as a gatekeeper system, for philosophical reasons.

Limited Options

As stated above, price is largely a function of:

- delivery system design;

PATRICK J. CAROLAN, M.D., of Bridgeport is President of CSMS-IPA. VINCENT J. CATRINI, M.D., of Middletown is Chairman of the Board of M.D. Health Plan, Inc.

- practice/referral patterns of caregivers;
- component cost(s).

Therefore, if M.D. Health Plan is to compete in price and give physicians what they believe is fair compensation for their efforts, the flexibility must come from one of the following:

a. Restrict utilization by creating barriers to patient access—cost will decrease as utilization decreases. The plan will be competitive and M.D. Health Plan can fully pay physicians' charges. Concomitantly, decreased utilization will decrease patient volume in the physician's office.
b. Restructure physician/patient care patterns—recognize that there exist competing non-M.D. services (eg, hospitals, laboratories, etc.) among our health care delivery system network. Reward those more efficient through increased service volume to those facilities. With increased volume you will receive increased discounts. Savings from volume discounts can be used to pay fully physicians' charges. Physicians will need to change referral patterns and use selected facilities for MDHP patients. Redirecting these referrals may be inconvenient for some physicians.
c. Increase physicians' financial risk. Increase the withhold portion of fees when utilization exceeds budget. Enable physicians to continue to practice as usual but physicians' total reimbursement is reduced until utilization comes in line with budget.
d. Empower doctors locally with greater involvement in the economics of their patient care, giving them their practice patterns on cost, and identifying favorable and unfavorable performance. Exercise control over the non-M.D. component enabling doctors to select "cooperative" services and facilities consistent with their cost management objectives.

Clearly, something has to change. In the short run, the CSMS-IPA is forced to do (c) but desires to do (d). The first two, (a) and (b), currently are incompatible with either our philosophy or structure. However, elements of both will continue to be studied if positive aspects can be defined while minimizing the negative impact on patient care access and physicians' practice patterns. But it may come to this:

> Either physicians take control of their destiny or be relegated to becoming employees, working for someone whose professional interest and objectives may be in conflict with their own.

Solution

M.D. Health Plan is the solution, but before there is gain will come pain. Let us discuss the pain first.

It is unrealistic and noncompetitive to expect any participating physician to resign from other HMOs or managed care companies. Therefore, M.D. Health Plan and the CSMS-IPA will need to do that which is necessary to survive financially. This will mean that periodically, M.D. Health Plan and CSMS-IPA, as now, request physicians to sacrifice short-term economic gains. Not unlike new start-ups in other competitive industries, the owners or benefactors appropriately assume the brunt of economic downturn. If this creates undue hardship for any physician, consideration could be given to such individuals. In general, M.D. Health Plan's patients will not represent a significant portion of any single physician's practice and, therefore, a temporary 10% increase to withhold or 4% when annualized should have minimal effect.

Here is the gain: physicians in Connecticut will remain in control of the way care is rendered to their patients. Physicians will define quality and work in "partnership" with employers who finance care to ensure the proper balance between cost, access, quality, and comprehensive care. Additionally, competing managed-care companies will be held to a physician-defined standard of care that, if not duplicated, will raise employer concerns about their efficacy and the quality of their health service delivery system. Having the physicians' HMO will keep other HMOs in check when dealing with physicians. If physicians have their alternative in the market, they will always have a bargaining chip with competitors.

To accomplish this, physicians must accept the premise that no one other than physicians will balance the medical needs of their patients while considering the economic implications of their actions. Most physicians are compelled by their own ethics to maintain the proper balance.

Skeptics suggest the structure of the medical care system is at fault. The fee-for-service payment structure, the imbalance (cost-shifting) between government and private sector reimbursement, the oversupply of doctors, and consumers' inability to make informed choices impede the cost-effective delivery of health care.

Placing this blame on physicians is oversimplifying the health care cost dilemma. Many forces beyond physicians' control contribute to the problem:

- new, higher cost technology;
- inflation;
- rising costs to non-M.D. component of medical cost structure;
- consumer expectations;
- aging population;
- consumer environmental and behavioral habits;
- malpractice insurance rates;
- antitrust regulations.

Managing Care Not Cost

Removing control over the practice of medicine from physicians will not solve the health care cost dilemma.

Motivating physicians to adopt acceptable practice standards is eminently more acceptable to physicians and will be far more effective in the long term.

However, if physicians are to retain control over their destiny, they must accept the cost-containment objective that managed care seeks to achieve, and acknowledge that business-as-usual medicine will no longer work. As long as physicians control allocation of medical resources they will always be viewed as having the last word. In other words, once patients gain access to medical care, the physician determines, when, where, how much, and for how long care is provided.

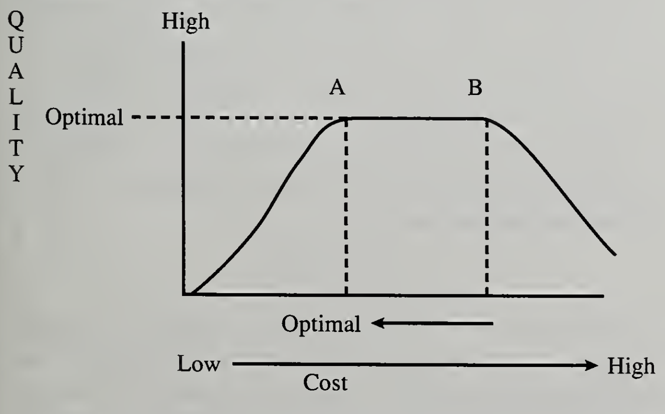
When the Connecticut State Medical Society developed its HMO in 1987, physicians' expectations were raised and it was held to a different standard from other HMOs. Specifically, physicians' desired:

- no intervention in their practice;
- ability to use existing referral patterns;
- open access to their patients.

Physicians claimed that:

- I practice cost effectively;
- my practice includes sicker patients, that's why my costs are higher;
- litigating conditions require me to practice defensively and that's why I order more tests;
- my practice requires me to do that procedure in the hospital;
- I need higher fees because my overhead is greater;
- I need higher fees because I do more procedures in office.

When taken in combination, these claims suggest that some doctors desire business-as-usual health care, not an HMO. Their points may be valid, but competitive market conditions will not allow M.D. Health Plan to operate business-as-usual health care. Very few physicians render inappropriate care; however, many physicians could practice more cost effectively. M.D. Health Plan and CSMS-IPA's challenge will be to motivate physicians in that direction. Graphically, this can be illustrated as follows:



The graph shows at point A the physician, in his treatment of patients, achieves maximum improvement of health status for the lowest dollars expended. Point B indicates achievement of the same level of health status but at a higher cost. Motivating physicians to practice more cost effectively, ie, moving from point B to point A means that they will give up some income because they will provide fewer services.

Most successful HMOs use structural limitations on their delivery system (ie, health care rationing) to achieve this end. Physicians will not accept this, particularly coming from a Medical Society sponsored HMO. Physicians must strategically view cost management not as an end in itself but as a stepping stone to utilization management.

It would be of greater benefit to reward physicians for cost-effective care than for lowest cost care. However, this will be difficult unless physicians accept their role as "captain of the ship" and exert influence over hospitals and other providers to cooperate with their HMO's case-management practices. It is in this role that physicians can retain control over the practice of medicine while achieving the cost savings sought by payers and employers.

M.D. Health Plan's Approach

Instead of being a mechanism to ration care, our medical management systems will need to build financial incentives that encourage physicians to use the most cost-effective levels of care. For this to work will require bringing local physicians into closer alignment with the HMO about what constitutes acceptable practice standards. This is eminently more difficult to achieve because, if required, all doctors will need to be in lock step with their HMO's objective. The larger the physician network the harder it is to get results. How we structure our network and use incentives and disincentives to motivate physicians' behavior will be extremely important.

Local Physician Empowerment

This new approach to medical management will effect physicians on two dimensions:

- encouraging greater physician involvement in the economics of patient care by creating smaller local physician groups that deliver care in a homogeneous medical service area;
- further refine the statistically based medical review methodology comparing treatment patterns for different diagnoses against acceptable norms and comparing physicians' medical cost patterns to peers.

M.D. Health Plan/CSMS-IPA is introducing this concept which is intended to encourage greater physician involvement in the economics of patient care. The system creates a series of small local IPA chapters (LCIPA) comprising of physicians affiliated with a designated hospital(s). This grouping of physicians with similar practice patterns recognizes that health care is provided through a distinct network of hospital affiliated professionals.

There are many incentives for physicians to join a LCIPA. One immediate benefit is that the HMO has linked utilization costs (adjusted according to case mix) with the LCIPA physician's potential withhold return. In this way physicians who manage their patients' care efficiently may receive a larger percentage of their withhold return at the end of the year. More careful management translates over time to competitive premiums for HMO members and prospective members. Offering competitive premiums will maintain, and perhaps improve, MDHP's posture in the marketplace. Finally, the arrangement preserves the independent practice concept because the physicians retain control over the practice patterns of a small network of their colleagues.

The LCIPA system is consistent with MDHP's mission to manage the health care needs of its members with high quality, reasonably priced benefits and programs, to promote and provide wellness and prevention opportunities, and, most important of all, to encourage a good patient-physician relationship. The goal of the arrangement is to place the responsibility, and the reward, for both quality and cost exactly where it should be—with the physician. Physicians are compelled by their own ethics to maintain quality. The HMO will provide them with the management tools and the administrative support they need to control costs.

The LCIPA concept encourages physicians to make slight changes in their practice patterns that do not compromise quality but have great effect on cost. Some of these decisions might involve prescribing generic drugs of equal efficacy for brand names, referring patients to laboratories with lower charges, or using home-care services when appropriate.

Here is how the system is designed to work:

First, a LCIPA is created. From that group, a physician coordinator, who will receive specialized training in practice management, is selected as a liaison between the LCIPA physicians and the HMO. The LCIPA creates its own utilization review and quality assurance advisory committee to provide support for the physician coordinator and to assist in the review and analysis of utilization data. Funding for these functions will be provided by the CSMS-IPA. Existing CSMS-IPA utilization review and quality assurance guidelines will remain intact.

MDHP will provide the LCIPA with administrative support through the medical director, and designated representatives from the Health Services, Professional Relations, and Operational Departments. Perhaps most importantly, MDHP will share valuable data relating to physician practice patterns. This data will track and compare costs associated with inpatient and outpatient visits, services ordered, and admissions. At a glance, physicians can identify the time and money devoted to each area of their practice. This information is intended to be viewed as a management tool and is, of course, confidential.

The factor that links utilization to the withhold return is the claims ratio. When a physician joins a LCIPA, MDHP will provide patient panel reports on a regular basis. At year end, the case-mix adjusted premium revenue will be divided into its actual claims experience to determine the group's claims ratio. Physicians whose LCIPA claims ratio is lower than the claims ratio for all CSMS-IPA would be rewarded with a potentially greater risk fund return. Recognizing the key role hospitals play in this scenario, MDHP will develop an incentive formula with the hospital to ensure its active partnership in the arrangement.

Statistically Based Medical Review Methodology

M.D. Health Plan will use a special data system to track the LCIPA activity. It represents state-of-the-art management of ambulatory care services using a statistically based review methodology that compares treatment patterns for different diagnoses against acceptable standards. The standards are based on national practice norms, modified for Connecticut practice patterns and adjusted for severity of illness, age, sex, and dual or multiple diagnoses. This system reviews each physician's claim to ensure acceptable practice patterns.

Resulting profiles are developed for each physician comparing his or her practice patterns for similar diagnosis with colleagues. The data are shared with the physicians in an attempt to modify their future behavior.

Profiling physicians will occur on two dimensions: comparison of treatment patterns to preestablished norms and comparison of resource utilization (cost) patterns relative to peers in similar specialties.

Both underutilization and overutilization will be identified. Physicians with favorable patterns will be rewarded through reduction or elimination of withhold and will share in any LCIPA surplus in hospital reserves. Conversely, physicians whose practice continues to exceed acceptable levels will be penalized through increases in withhold levels. All data identifying physician outliers will be shared with the physician, and sufficient time will be given to allow practice patterns to come within acceptable norms.

Conclusion

The technical know-how is now available to make managed care work effectively at M.D. Health Plan. Getting hospitals, physicians, and other caregivers to commit to making it work is the strategic challenge. Overcoming the notion that managed care alters the decision making of physicians and hospitals by interjecting a complex system of administrative procedure will be critical if we are to meet this challenge.

Effective managed care delivery systems control quality and use of services as well as clinical cost and operational expenses. However, as all physicians can attest, the term "managed care" is not necessarily applicable to all Connecticut HMOs. It depends on the extent to which essential building blocks of managed care (for example, benefit design incentives, utilization management procedures, quality assurance protocols, and information system capability) are integrated into program operations, and on whether performance standards are achieved.

As M.D. Health Plan continues to refine and enhance its health services delivery system, we ask physicians to be aware of how their decisions effect cost and quality. In a competitive managed care market, quality care will be practiced efficiently, and inefficient plans will not survive over the long term.

"I'm practicing medicine the way I think it should be practiced, sans the paperwork and administrative overload."

Owen Brodie, MD, joined CompHealth's locum tenens medical staff in 1989, after 21 years in private practice. Since then he's worked in temporary assignments in state facilities, filled in for attending physicians, covered for private practitioners across the country.

A pilot. A historian. A board-certified psychiatrist. Southern to a fault. Owen Brodie knows...

It's a great way to practice medicine

CompHealth
LOCUM TENENS

1-800-453-3030
Salt Lake City ■ Atlanta ■ Grand Rapids, Mich.

MATERIAL printed in *Connecticut Medicine*, the Journal of the Connecticut State Medical Society, is covered by copyright. Written permission must be obtained before reproducing, in part or in whole, any material published in *Connecticut Medicine*.

Connecticut Medicine does not hold itself responsible for statements made by any contributor.

PROSPECTIVE authors preparing manuscripts for submission to *Connecticut Medicine* should consult "Information for Authors." This material may be obtained from the *Journal* office. The manuscript, including references, should be typed double spaced; all material, including figures, should be submitted in duplicate. Manuscripts submitted on disk are encouraged. The transmittal letter should designate one author as correspondent and include his complete address and telephone number.

MANUSCRIPTS should be submitted to the Editor, Robert U. Massey, M.D., *Connecticut Medicine*, 160 St. Ronan Street, New Haven, CT 06511. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication.

ALTHOUGH all advertising material is expected to conform to ethical medical standards, acceptance does not imply endorsement by this journal.

REPRINTS are available at an established schedule of costs. A reprint rate and order form is sent to the author with his page proofs.

SUBSCRIPTION RATE: The subscription price per year for members of the Connecticut State Medical Society is included in the annual membership dues. The subscription price per year for nonmembers is $25.00 (Canada and foreign, $40.00). Single copies $4.50.

ADDRESS editorial and business correspondence to: *Connecticut Medicine*, 160 St. Ronan Street, New Haven, CT 06511. Telephone (203) 865-0587; Fax. (203) 865-4997.

CONNECTICUT MEDICINE is the official publication of the Connecticut State Medical Society, a voluntary non-profit association organized for scientific and educational purposes, published monthly for members of the Connecticut State Medical Society.

CALL FOR PAPERS

Members of the Connecticut State Medical Society reading papers before other organizations are requested to submit their papers to the JOURNAL for consideration by the Board of Editors for publication. Authors preparing manuscripts for submission to *Connecticut Medicine* should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office. Adherence to the instructions will prevent delays both in acceptance and in publication. Papers that have been set on a computer with foppy disk capabilities (3-1/2 or 5-1/4) should be submitted on disk along with the hardcopy andindication as to which wordprocessing program was used.

Please send them to:

Robert U. Massey, M.D.
Connecticut Medicine
160 St. Ronan Street
New Haven, CT 06511

INQUIRE TODAY.

ENDORSED BY
THE CONNECTICUT STATE MEDICAL SOCIETY

—OFFERING—

- **Hospital, Surgical and Major Medical Insurance**
- **Term Life Insurance**
- **Employee Benefit Package**

—FEATURING—

- **OFFICE OVERHEAD AND DISABILITY INSURANCE PACKAGE**

[Rates based on a 30 day waiting period for ages 30-39]

$1500.00 weekly disability indemnity
semi-annual premium is $528.15*

$20,000.00 monthly overhead indemnity
semi-annual premium is $561.00

* Optional Residual and COLA benefits available

ayr

AYR INSURANCE AGENCY

105 SANFORD STREET HAMDEN, CT 06514 288-4302

Improving Health-care Costs with Minimal Compromise

DICKERMAN HOLLISTER, JR., M.D.

EVERY day we hear about our country's health care crisis.

We are spending too much for medical care, now up to 12% of the GNP—more than any other nation. And despite this, we still have 33 million Americans whose lack of health insurance limits the care they receive.

How did our medical system, which arguably produces the finest care in the world, get into such a financial mess? How can we account for a doubling of health expenditures in only the last 10 years?

About half the decade's rise in health care expenditures is due to changes outside the medical system. The most important of these are national inflation, growth of the U.S. population, and the aging of our citizens. No type of health care reform will change these economic and demographic realities.

The other 50% is due to the growing intensity of medical care: More specialists, more treatments, and more tests. This intensity has three causes. First is scientific development. From transplant surgery to gene therapy, from laser scalpels to hip replacements, medicine has been advancing at a fantastic pace. The results are better. Our declining infant mortality rates and growing elderly population prove it. But the costs are greater. How could they not be?

The second reason for the intensity of today's medicine is our changing society. More Americans than ever now live in poverty, and poor people require expensive medical treatment. For examplé, the poor seldom receive simple, economical treatment for high blood pressure. They are neglected until kidney failure results.Then they receive expensive kidney dialysis—intensive care. The same is true for teenage mothers, many of whom have premature infants. It costs $2,000 per day to treat these preemies, but only $500 per pregnancy to provide the prenatal care to prevent premature birth (and even less for family planning services). These people do not receive cost-efficient, preventive care because budget deficits prevent adequate funding of welfare and medicaid. Our social programs are penny-wise leaving our medical system pound-foolish.

Other changes in our society lead to rising health care costs. Drug and alcohol abuse not only lead to health problems for the abuser but for innocents as well from motor vehicle accidents and domestic violence. We've made the possession of handguns a constitutional right. While not long ago adolescents settled their differences with fists, needing at most a few sutures in the emergency room, now they come in with multiple gunshot wounds, requiring weeks of intensive care at no small expense. And, of course, we now have AIDS. Roughly 1.5 million Americans are infected with this virus; virtually all will need expensive care by the decade's end.

Third, unique "American factors" contribute to the intensity of today's health care. Our patients demand the most advanced and sophisticated technology, knowing that their insurance insulates them from the direct costs of such care. Daily we doctors hear, "I want the best treatment possible, no matter what it costs. My insurance will pay." It's very different in other countries. In Sweden, for example, an 80-year-old with a heart attack does not go to the coronary unit, no matter how much he or his family wishes. Sweden's socialized insurance program knows that such intensive treatment is not "cost-effective" for the elderly. But in the U.S. our seniors do get this care. Whether that is right or wrong is a matter of opinion. What is fact, however, is that it would be virtually impossible to change the singularly American view that all receive first-rate care on demand.

Physicians also suffer from "American factors." We want the best for our patients and have literally sworn to provide just that. But we also directly benefit from such services, and the more we provide the more we make. Finally, we fear that if we do not provide all services, we will personally be penalized by the malpractice system. In no other country is the tort system so punitive.

How then can we control our country's health care costs, maintain its quality care, and simultaneously expand access for our nation's uninsured? We must compromise. Neither we nor any other country can afford to provide all services to all people. Instead, we must strike

---

DICKERMAN HOLLISTER, JR., M.D., is president of the Fairfield County Medical Association and a practicing oncologist at Greenwich Hospital. Councilor of the Connecticut State Medical Society.

some balance between our current system, which provides all services to most people and a Canadian-type program, which provides most services to all people.

Let's look in more detail at the Canadian system. It has both pros and cons. Its pros are considerable—it boasts universal access to early care for preventive services. The absence of out-of-pocket expense encourages utilization of cost-effective treatment. The single, provincial payor requires less paperwork, so that money is spent on health care, not administration. Most importantly, the quality of medical care is very high. There is good value for the health dollar.

The cons are more subtle. First, there are waiting lists for nonemergency services. If you're 70 and need a cataract removed, you may wait a year. It's not life-threatening, but if you're that age, a year unable to read or watch television is a major hardship. The same is true for a hip replacement, one less year to walk. Even life-saving heart surgery has waiting lists, forcing many Canadians to come to the U.S. for their operations. Second, the technology may not be available. In all of Canada, there are only 12 MRI scanners. We have nearly 1,400. The months of delay for an MRI in Canada can be critical to the cancer patient. Third, the Canadian system is not designed for medical research or capital development. Canada has nothing comparable to our National Institutes of Health or Centers for Disease Control. In fact, Canadians depend on our system to spend dollars for scientific breakthroughs. If we adopt their system, upon whom will we depend? Finally, the Canadian system itself is now increasingly stressed by rapidly rising costs as Canadians start demanding the technology they see so readily available in the U.S. In fact, the rate of cost growth in the last decade has been greater in Canada than in the U.S.

If the Canadian system is not for us, what are the alternatives? The Pepper Commission, chaired by Senator Jay Rockefeller, proposed a package of health care reforms more suitable for our country. It recommended a "play or pay" model which combines private and public insurance programs. A similar plan, requiring more on private coverage, has been designed by physicians of the American Medical Association.

While these proposals have substantial differences, their common theme is to build on and improve our current health care system. Despite its flaws, our system provides excellent care and gives both patient and physician more choice, flexibility, and responsibility than in any other nation. To improve access, these plans expand insurance coverage by utilizing the current employer-based method. Three-quarters of the uninsured are either workers or their dependents. If we could provide the appropriate tax incentives to encourage all employers to provide insurance, we would go a long way towards providing universal coverage. Most of the nonworking uninsured could then be covered by state risk pools and the expansion of medicaid benefits, which currently only cover 40% of those below the federal poverty level. Coupled with an expanded medicare program, nearly all citizens would be adequately covered.

These plans also remove many of the barriers to private insurance. Proposed legislation would prevent redlining of high risk groups, eliminate employer based "job lock," and restrict limitations on preexisting illness.

Physicians are a major part of the reform. We have to save money. How? We've already started to emphasize preventive care and to educate our patients about the dangers of smoking, excessive drinking, and sedentary living. We are developing "practice parameters," standards of care that are physician-designed protocols for treating complex illnesses. Their goal is to reduce futile and expensive care while still providing our patients with appropriate treatment and technology. Many of these protocols are in use today.

Liability laws must be changed so that settlements are just and awarded to the patient, not eaten up by the legal system. Defensive medicine costs would decline if physicians felt the system were just. Finally, administrative costs must be reduced by simplifying and standardizing insurance and reimbursement procedures.

These reforms would provide uniform care to all with minimal compromise. While there would, at least in the short term, be some additional costs, they would be far less than establishing an entirely new federal system. Such a Canadian approach would cost us billions in new taxes to create the bureaucratic machinery. Not only would that be expensive, it is politically unrealistic.

The recent debacle over catastrophic health insurance tells us that Americans will not accept higher personal tax rates to improve medical care for the disadvantaged. Finally, ask yourself if our federal government is up to the task of regulating all health care. How well does Congress control expenditures now? And do we want queues in our operating rooms like those in the post office?

There is no easy solution to our country's health care problems. We simply cannot provide everything to everyone; some compromises must be made. The trick is to make them deliberate and incremental, so that we do not go from an imperfect system to a disastrous one. Let us improve our system, not abandon it. By the reforms outlined above, we can best balance the competing goals of expanding access, reducing costs, and maintaining quality. The system won't be perfect, but it can be the world's best. We should work for no less.

The Education of a Pathologist

TSUYOSHI INOSHITA, M.D.

HERE I was, a pathologist in mid-life crisis. Every goal I had been aiming for seemed to crumble in front of me. I had always been the sort of person who preferred independence and freedom from external restrictions, and thought, as a naive medical student, that pathology would give me such freedom. I would be free of troublesome human relations. But pathology turned out to be suffocating, sterile. I felt constrained. After a few attempts as an attending pathologist in various institutions, I finally returned to clinical medicine. Imagine, a medical resident in my mid-thirties! And here I was going back to clinical medicine when so many seemed to be leaving it. I would practice somewhere I dreamed, as a general internist, away from the politics of institutions.

This was arduous, this abrupt transition in my life. My nine-to-five existence was suddenly transformed into interminable 36-hour shifts. I had no days off. It seemed as though I lived in the hospital. I counted the days until the end of that first year. My life gradually improved and I found time to think. I decided to give medical oncology a try. It would be, I thought, the ideal blend of my interest in cancer and my newfound vocation of patient care. I applied for and accepted a fellowship in medical oncology.

Nor was this easy. I was a fellow approaching forty years of age. And on the very first day of my fellowship, I was assigned to the outpatient clinic and saw a small elderly lady in her early seventies. Her name was Mrs. E, and she had just been diagnosed with an extensive small cell carcinoma of the lung. She came with her husband. Their only son, a family practitioner, lived in another large city hundreds of miles away. He never visited his parents anymore. The patient and her husband lived alone, each having the other only for support. Mrs. E was an endearing little lady, and despite her tragic situation, she was stoic and accepting. Her husband was the joke-cracking, jovial type but he couldn't hide his nervousness. I discussed with them the possibility of a good immediate response of her tumor to chemotherapy, but the nevertheless dismal prognosis for long-term survival. Mrs. E agreed to a trial of chemotherapy.

She responded to the medicines quite well. As the treatment progressed I became good friends with both Mrs. E and her husband. They always came together, her husband driving the 45-minute journey from their home, and they were always in the examination room together, smiling, waiting for me. He would tell a few jokes and then leave the room. It was always the same. Mrs. E told me that he was going outside of our smoke-free building to have a cigarette. She didn't confide in me that while she didn't mind dying, she worried about her husband. After six cycles of chemotherapy, she gained complete remission. They were a happy couple again, she cooking for him and he playing golf, cracking his jokes.

Some months later I received a call from an emergency room physician. He told me that Mrs. E was in his department, having been brought in after a seizure, and that her CT scan showed brain metastases. He wanted to transfer the patient to my hospital. When I saw Mrs. E, I was surprised to find how much older she looked, how much more frail. We gave her brain irradiation and started a second chemotherapy protocol. Miraculously she went into a second complete remission. But she knew that this also would be transient, that the inevitable was coming.

"Doctor, I know you can't cure me. Still I worry about my husband. I don't think he can cope without me." I didn't have any words for her, there was nothing I could say.

The encounter became more difficult. Mrs. E and her husband came in with a camera and she took pictures of me. I tried to smile, but knowing the inevitable, fought back the tears. And a few months later, the second recurrence came back as we knew it would and I had to transfer her to a hospice. It was only a few weeks later that I received a call from her husband and was told that

---

TSUYOSHI INOSHITA, M.D., *Ann Intern Med* 1992;117:608.

Requests for Reprints: Tsuyoshi Inoshita, M.D., Portsmouth Cancer Center, 1745 27th Street, Portsmouth, OH 45662.

HEALTH SCIENCES LIBRARY

Mrs. E had died peacefully at home. I hung up the phone and began immediately writing a letter to Mr. E, telling him of his wife's concerns for his welfare. He responded, grateful for my care of his wife, for my involvement with them and for my communication with him. How bittersweet was this connection and how difficult!

The fellowship is over. The lessons are not. I am a solo oncologist practicing in a small town. There are many Mrs. E's, many spouses similar to hers. Frequently I connect, and too frequently find myself crying inside. But I've not regretted my choice, and feel fulfilled, finally having become a doctor.

MOVING? ADDRESS CORRECTION?

Attach your current mailing label here
and enter correction below.
Please give us six weeks notice.

Name ____________________

Address ____________________

City ____________________

State ____________ Zip ________

Send address changes to:

Connecticut State Medical Society
Membership Department
160 St. Ronan Street
New Haven, CT 06511

PHYSICIANS ! DENTISTS !

Your office may qualify for a *FREE* Practice Management Computer System.

Medical Records ■ Scheduling ■ Patient Accounting
Electronic Claims

	Normal Price	Special (if qualified)
Med-Ease Software	$6,000	$ 0
386-33 comp. system	6,500	0
Software support	1,500/yr.	0
Hardware support	1,080/yr.	0

For more information or demonstration, call **393-2000**

DAGAR **BETHANY, CT**

THE FUTURE IN HEALTHCARE SOFTWARE

DRUG INFORMATION UPDATE: HARTFORD HOSPITAL

Stress-Related Gastritis: Focus on the Use of H_2-receptor Antagonists and Antacids

ELIZABETH CHIME-UDEH, PHARM.D.

Introduction

STRESS-related gastritis previously associated with severe traumatic injury is common in nearly all critically ill patients.[1-4] Endoscopic evaluation of various categories of critically ill patients show that 74% to 100% have stress-related mucosal damage (SRMD) within 72 hours of onset of major illness.[1-4] Though the incidence of bleeding from SRMD is about 5-20% the mortality rate from overt bleeding has been shown to be 64-100%.[2,3,5,6] In recent years there has been significant decrease in the incidence of bleeding from SRMD; however, there is no evidence of reduced incidence of SRMD.[7-8] The development of stress-related gastritis involves multiple physiologic derangement; the actual pathophysiologic mechanism is not well understood, but is believed to be due to an imbalance between inherent mucosal protection and destructive properties of the gastric acid.[9-15] The universal awareness of the morbidity associated with bleeding from SRMD warrants the use of various pharmacologic agents (antacids, H_2-blockers, cytoprotectives, prostaglandin analogue, pirenzipines) in an effort to prevent or treat it. This review focuses on the rationale for medical prophylaxis of SRMD and appropriate use of antacid and H_2-receptor blockers for this indication.

Rationale for Medical Prophylaxis

A number of studies, including those that demonstrate a 64-100% mortality from overt bleeding, have established the definite need for prophylaxis of SRMD in critically ill patients.[16-20] Consequences of overt bleeding, such as decreased hematocrit and hypovolemia, further complicate the patient's illness and increase the patient's time spent in the intensive care unit (ICU). Blood transfusions are often required and are not without risk. Post-transfusion hepatitis occurs in 7-10% of patients receiving transfusions, with 40-60% of them developing chronic hepatitis.[18] Surgical intervention, usually indicated when medications fail to control overt bleeding, is expensive and has its own intrinsic risks.[17,18,21] Finally, preventive treatment with appropriate medications is easier, effective, and less costly than treatment of bleeding.[5]

Prophylaxis of SRMD

The goal of SRMD prophylaxis is to identify patients at high risk and institute preventive therapy with one of the available and effective agents. Intragastric $pH > 4$ has been associated with decreased frequency of bleeding from stress-related gastritis. Thus, agents such as H_2-receptor antagonists and antacids that neutralize or decrease gastric acid secretions are used for prophylaxis. Recently, prophylactic efforts have also focused on cytoprotection with agents such as sucralfate and prostaglandins to enhance mucosal defense.[5,9]

The H_2-receptor antagonists have been shown in controlled studies to improve gastric mucosal defense and to decrease the incidence or progression of SRMD in critically ill patients.[22,23] Several studies comparing both cimetidine and antacids to placebo showed that both have a significant advantage over placebo for prophylaxis against SRMD in critically ill patients.[23-26] In some earlier studies, investigators using intragastric pH control as a therapeutic end-point claimed that antacids were better than H_2-receptor antagonists for prophylaxis of stress related gastritis.[24,25] Most of the studies favoring antacids rather than cimetidine, compared every one to two hours antacid titration to every four to six hours cimetidine administration.[23-25] The problem with intermittent cimetidine administration, especially every six hours, is its short half-life and inconsistent pH control.[26] The result of a 1991 meta-analysis on stress ulcer prophylaxis in critically ill patients indicated that H_2-receptor antagonists are superior to antacids in prevention of clinically

---

ELIZABETH CHIME-UDEH, PHARM.D., Clinical Specialist, Medicine, Hartford Hospital; Assistant Clinical Professor, University of Connecticut, School of Pharmacy, Storrs.

This series on Drug Information Updates from Hartford Hospital are provided by the Drug Information Center, Department of Pharmacy Services, Hartford Hospital.

Editors: Moses Sing Sum Chow, Pharm.D. and Robert A. Quercia, M.S., R.Ph.

important bleeding.[27] This new finding is due to improved methods of evaluating SRMD and better study metholology.[23,27] Of the available H_2-receptor antagonists, cimetidine and ranitidine have been extensively evaluated for prevention of SRMD.[26-30] To date, cimetidine is the only agent approved by the FDA for prophylaxis of SRMD.

In an effort to achieve better pH control with cimetidine, continuous infusion (CI) over 24 hours was used in a number of trials. Tome et al, in a prospective study of 20 patients, observed that CI of cimetidine 2 g in a 50 mL solution over 24 hours maintained gastric pH between 4-7 in all study subjects and 67% of the time the pH was above 5.[26] Ostro et al, in his comparison of bolus *v* primed infusion in 23 patients noted that 1,200 mg of cimetidine per day as a CI could maintain intragastric $pH > 4$ in 20 of 23 patients, while the same total daily dose given as 300 mg boluses every six hours achieved the same result in only five patients.[29] Kingsley compared the effect of various methods of administering antacid and cimetidine for stress ulcer prophylaxis.[25] His investigation showed that CI was superior to the intermittent method for both cimetidine and antacid.[25] The newer H_2-receptor antagonists, ranitidine and famotidine, have a longer elimination half-life; thus intermittent administration of these agents at the recommended dosing interval is more effective than cimetidine. However, the CI of these H_2-receptor antagonists has been utilized and there may be cost advantages with this mode of administration.

Other agents that have been shown to be effective for prophylaxis of SRMD include sucralfate, prostaglandins, and the antimuscarinic agent pirenzipines.[23,27] Of all three agents, sucralfate has been better evaluated for this indication. As a result of weaknesses in study designs and data analysis, data are insufficient to determine the relative efficacy of sucralfate for prophylaxis of SRMD.[31-35]

Selection of Appropriate Agents

Certain factors need to be considered when choosing an agent or agents for stress ulcer prophylaxis.

Ease of Administration.—Most critically ill patients are unable to take medications orally. Thus, to use antacids for prophylaxis of SRMD will necessitate the use of a nasogastric. Effective antacid prophylaxis requires hourly[36] or every two hour[37] administration which is logistically difficult and takes up a great deal of ICU nursing time.[13] On the other hand, continuous intravenous infusion of H_2-receptor blockers is much easier especially when most ICU patients already have an IV access.[13,38]

Cost.—Continuous infusion of H_2-receptor blockers is cost effective. Both the preparation and administration takes place just once a day as opposed to preparing and administering multiple minibags. Antacid titration is labor intensive and may be more costly when nursing time is included in the total cost of antacid therapy.[39]

Safety.—The safety of the agents used for SRMD is important. Diarrhea seems to be the most common adverse effects with antacids; however, a significant number of patients experience regurgitation, electrolyte abnormalities, and persistent alkalosis secondary to high gastric output.[25,40-42] Thrombocytopenia, mental confusion, and disorientation are adverse effects usually associated with the H_2-receptor blockers.[24,40-42] However, overall, these effects rarely occur at the recommended dosage regimen.[5,24,40] Some investigators have expressed concern about pharmacological blockade of H_2-receptors in the heart and the resultant bradycardia, negative inotropic effects, and coronary vasoconstriction.[40] The clinical significance of these potential cardiac effects are not known.

Other issues of concern with the prophylaxis of SRMD is the theoretically high incidence of aspiration pneumonia in ICU patients.[31,42] This problem tends to be a greater concern in patients receiving antacids.[4]

Potential Drug Interaction.—Cimetidine is known to interact with different classes of drugs. The most common interactions that may be clinically significant in an ICU setting involve theophylline, anticonvulsants, antiarrhythmics, and warfarin.[5,13,43] The interactions with theophylline may be minimal with continuous infusion of cimetidine.[43] Antacids are not totally devoid of potential drug interactions; they may inhibit the absorption of certain orally administered medications including quinolones, glyburide, glibizide, and ketoconazole.[5]

Renal Failure.—Both cimetidine and antacids may present problems in patients with renal impairment. Aluminium containing antacids (Riopan®, Trigastril®, etc.) cause significant rise in serum aluminium concentrations in patients with renal impairment.[13] The side effects of cimetidine are more common in patients with renal impairment.

Recommendations for SRMD Prophylaxis

Extensive literature reviews and institutional experience show that prophylaxis for SRMD is safe and effective. At Hartford Hospital the following is recommended:

1. Critically ill patients with multiple risk factors for SRMD receive medical prophylaxis with an H_2-receptor antagonist or antacid. Cimetidine is currently the only H_2-receptor antagonist approved for SRMD prophylaxis but both ranitidine and famotidine have been used successfully for prophylaxis.
2. Cimetidine administered as 900-1200 mg by CI over 24 hours. (At our institution we developed a safe and cost-effective H_2-receptor antagonist pro-

gram with cimetidine as the preferred agent. The annual cost savings to the hospital was $145,557; the CI of cimetidine was necessary to realize these substantial savings).[44]

3. Famotidine (40 mg/day) or ranitidine (300 mg/day) administered by intermittent injection or by CI over 24 hours may be used as an alternative especially in patients who should not receive cimetidine because of a potentially significant drug interaction or renal impairment.
4. Alternatively antacids, and whenever possible antacids with high neutralizing capacity (Maalox TC®, Riopan®, etc.), may be administered at a dose of 30 mL every 1-2 hours.
5. Prophylaxis should be continued until patients improve sufficiently to institute oral feedings.

Conclusion

Most critically ill patients have or will develop stress gastritis. About 50% of these patients will develop endoscopic evidence of bleeding which may require blood transfusion or even surgical intervention. These potential complications can be prevented using noninvasive, low-risk medical prophylaxis with the use of H_2-receptor antagonists or antacids.

REFERENCES

1. Kitler ME, Hays A, Enterline JP, et al: Preventing postoperative acute bleeding of the upper part of the gastrointestinal tract. *Surg Gynecol Obstet* 1990;171:366-72.
2. Gonzalez ER: Pathophysiologic changes in the critically ill patient: Risk factors for ulceration and altered drug metabolsim. DICP, *Ann Pharmacother* 1990;24(Suppl):5-7.
3. Reusser P, Gyr K, Scheidegger D, et al: Prospective endoscopic study of stress erosions and ulcers in critically ill neurosurgical patients: Current incident and effect of acid-reducing prophylaxis. *Crit Care Med* 1990;18:270-4.
4. Lucas CE, Sugawa C, Riddle J, et al: Natural history and surgical dilemma of "stress" gastric bleeding. *Arch Surg* 1971;102:266-72.
5. Peura DA and Heine KJ: Prophylaxis of stress-related gastrointestinal mucosal damage: Rational and options. *Hosp Formul* 1991;26(Suppl D):8-12.
6. Gottlieb JE, Menashe PI, and Cruz E: Gastrointestinal complications in critically ill patients: The intensivists' overview. *Am J Gastroenterol* 1986;81:227-38.
7. Ruiz-Santana S, Ortiz E, Gonzalez B, et al: Stress-induced gastroduodenal lesions and total parenteral nutrition in critically ill patients: Frequency, complications and the value of prophylactic treatment. A prospective, randomized study. *Crit Care Med* 1991;19:887-91.
8. Konopad E and Noseworthy T: Stress ulceration: A serious complication in critically ill patients. *Heart Lung* 1988;17:339-48.
9. Kleiman RL, Adair CG, and Ephgrave KS: Stress ulcers: Current understanding of pathogenesis and prophylaxis. *Drug Intell Clin Pharm* 1988;22:452-60.
10. Bruegge WFV and Peura DA: Stress-related mucosal damage: Review of drug therapy. *J Clin Gastroenterol* 1990;12(Suppl 2):S35-40.
11. Peura DA: Stress-related mucosal damage: An overview. *Am J Med* 1987;83(Suppl 6A):3-7.
12. Levinson MJ: Gastric stress ulcers. *Hosp Practice* 1989;March 30:59-68.
13. Geus WP and Lamers CBHW: Preventions of stress ulcer bleeding: A review. *Scand J Gastroenterol* 1990;25(Suppl 178):32-41.
14. Bank S: Stress ulcers-prevention of gastrointestinal bleeding in critical care units. *Med J Aust* 1985;142(Suppl 2):S17-20.
15. Martin LF, Max MH, and Polk HC, Jr: Failure of gastric pH control by antacids or cimetidine in the critically ill: A valid sign of sepsis. *Surgery* 1980;88(:59-68.
16. Miller TA: Mechanisms of stress-related mucosal damage. *Am J Med* 1987;83(Suppl 6A):8-14.
17. Shuman RB, Schuster DP, and Zuckerman GR: Prophylactic therapy for stress ulcer bleeding: A reappraisal. *Ann Intern Med* 1987;106:562-67.
18. Wilcox CM and Spenny JG: Stress ulcer prophylaxis in medical patients: Who, What, and How Much? *Am J Gastroenteral* 1988;83:1199-211.
19. Schuster DP, Rowley H, Feinstein S, et al: Prospective evaluation of the risk of upper gastrointestinal bleeding after admission to a medical intensive care unit. *Am J Med* 1984;76:623-30.
20. Dellinger RP: Pathophysiology, monitoring, and management of the ventilatordependent patient: Considerations for drug therapy, emphasis on stress ulcer prophylaxis. DICP, *Ann Pharmacother* 1990;24(Suppl):S8-11.
21. Zuckerman GR, Cort D, and Shuman RB: Stress ulcer syndrome. *J Intensive Care Med* 1988;3:21-31.
22. Zuckerman GR and Shuman R: Therapeutic goals and treatment options for prevention of stress ulcer syndrome. *Am J Med* 1987;83(Suppl 6A):29-35.
23. Tryba M: Prophylaxis of stress ulcer bleeding: A meta-analysis. *J Clin Gastroenterol* 1991;13(Suppl 2):S44-55.
24. Gonzalez ER and Morkunas AR: Prophylaxis of stress ulcers: Antacid titration *v* histamine H_2-receptor blockade. *Drug Intell Clin Pharm* 1985;19:807-11.
25. Kingsley AN: Prophylaxis for acute stress ulcers. Antacids or cimetidine. *Am Surg* 1985;51:545-47.
26. Tome G, Fiasse R, Reynaert M, et al: Effect of a 2 g cimetidine infusion on twenty-four-hour intragastric pH in critically ill patients. *Intensive Care Med* 1988;14:379-83.
27. Cook DJ, Witt LG, Cook RJ, and Guyatt GH: Stress ulcer prophylaxis in the critically ill. A meta-analysis. *Am J Med* 1991:91:519-27.
28. Lamothe PH, Rao E, Serra AJ, et al: Comparative efficacy of cimetidine, famotidine, ranitidine, and mylanta in postoperative stress ulcers. *Gastroenterol* 1991;100:1515-20.
29. Ostro MJ, Russell JA, Soldin SJ, et al: Control of gastric pH with cimetidine boluses versus primed infusions. *Gastroenterol* 1985;89:532-7.
30. Khan F, Parekh A, Patel S, et al: Results of gastric neutralization with hourly antacids and cimetidine in 320 intubated patients with respiratory failure. *Chest* 1981;79:409-12.
31. Tryba M: Sucralfate versus antacids on H_2-antagonists for stress ulcer prophylaxis: A meta-analysis on efficacy and pneumonia rate. *Crit Care Med* 1991;19:942-9.
32. Cannon LA, Heiselman D, Gardner W, Jones J: Prophylaxis of upper gastrointestinal tract bleeding in mechanically ventilated patients. A randomized study comparing the efficacy of sucralfate, cimetidine, and antacids. *Arch Intern Med* 1987;147:2101-6.
33. Tryba M, Zevounou F, Torok M, Zenz M: Prevention of acute stress bleeding with sucralfate, antacid, or cimetidine: A controlled study with pirenzepine as a basic medication. *Am J Med* 1985;79(Suppl 2C):55-61.
34. Driks MR, Craven DE, Celli BR, et al: Nosocomial pneumonia in intubated patients given sucralfate as compared with

antacids or histamine type 2 blockers. *N Engl J Med* 1987;317:1376-82.

35. Ryan P, Dawson J, Teres D, Navab F: Continuous infusion of cimetidine *v* sucralfate: Incidence of pneumonia and bleeding compared (Abstr). *Crit Care Med* 1990;18:S253.
36. Hasting PR, Skillman JJ, Bushnell LS, Silen W: Antacid titration in the prevention of acute gastrointestinal bleeding. *N Engl J Med* 1978;298:10415.
37. Zinner MJ, Zuidema GD, Smith PL, Mignosa M: The prevention of upper gastrointestinal tract bleeding in patients in an intensive care unit. *Surg Gynecol Obstet* 1981;153:214-20.
38. Greene WL, Bollinger RR: Cimetidine for stress-ulcer prophylaxis. *Crit Care Med* 1984;12:571-5.
39. Moore JG: Achieving pH control in the critically ill patient: The role of continuous infusion of H_2-receptor antagonists. DICP, *Ann Pharmacother* 1990;24(Suppl):S28-30.
40. Peura Da, Johnson LF: Cimetidine for prevention and treatment of gastroduodenal mucosal lesions in patients in an intensive care unit. *Ann Intern Med* 1985;103:173-7.
41. Tryba M: Side effects of stress bleeding prophylaxis. *Am J Med* 1989;86(Suppl 6A):85-93.
42. Weigelt JA, Aurbakken CM, Gewertz BL, Snyder WH III: Cimetidine *v* antacid in prophylaxis for stress ulceration. *Arch Surg* 1981;116:597-601.
43. Strembo MA and Karlstadt R: H_2 blockers in stress ulcer prophylaxis (letter). *Hosp Practice* 1989;Oct 30:17, 20.
44. Quercia RA, Chow MSS, Jay GT, Quintiliani R: Strategy for developing a safe and cost-effective H_2-receptor antagonist program. *Hosp Formul* 1991;26(Suppl D):20-24.

CALL FOR PAPERS

Members of the Connecticut State Medical Society reading papers before other organizations are requested to submit their papers to the JOURNAL for consideration by the Board of Editors for publication. Authors preparing manuscripts for submission to *Connecticut Medicine* should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office. Adherence to the instructions will prevent delays both in acceptance and in publication. Papers that have been set on a computer with foppy disk capabilities (3-1/2 or 5-1/4) should be submitted on disk along with the hardcopy andindication as to which wordprocessing program was used.

Please send them to:
Robert U. Massey, M.D.
Connecticut Medicine
160 St. Ronan Street
New Haven, CT 06511

Important Advances in Clinical Medicine

Radiology

The Scientific Board of the California Medical Association presents the following inventory of items of progress in radiology. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome, and an authoritative reference both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, researchers, or scholars to stay abreast of these items of progress in radiology that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Radiology of the California Medical Association, and the summaries were prepared under its direction.

Magnetic Resonance Imaging of Musculoskeletal Tumors

PRIMARY and metastatic musculoskeletal tumors are common and potentially life-threatening. Although conventional radiographs are the primary means of detecting and diagnosing these tumors, plain films have two shortcomings—poor definition of tumor extension and poor detection of early osseous metastases. Magnetic resonance imaging (MRI), because it can produce images in multiple planes while displaying superior soft-tissue contrast resolution, has had a substantial effect on the description, preoperative staging, and postoperative follow-up of musculoskeletal tumors.

Several MRI findings increase a physician's index of suspicion for malignant tumor: inhomogeneous signal intensity, irregular margination, osseous destruction, encasement of neurovascular structures, and disruption of more than one compartment of bone, joint, or muscle. As a general rule, osseous and soft-tissue tumors have low or intermediate signal intensity on T1-weighted images and a high signal on T2-weighted images. Osteoblastic lesions are often the exception; they have low signal intensity on both T1- and T2-weighted images. Tumor conspicuity and delineation are usually not improved by administering intravenous gadolinium—gadolinium enhancement actually may obscure tumor margins.

The staging of musculoskeletal tumors requires determining the tumor extent, the histologic grade, and the presence or absence of metastases. A bone scan is the method of choice when screening the whole body for skeletal metastases, but its findings are notoriously nonspecific and its spatial resolution is so poor that precise preoperative planning is precluded. Computed tomography (CT), on the other hand, has good spatial resolution and effectively detects early cortical erosions, but its contrast resolution is inferior to that of MRI. The present consensus is that MRI is superior to CT for defining the infiltration of the tumor into bones, muscles, joints, and neurovascular structures. Tumor margins visualized by MRI have been shown to match closely those found by pathologic examination. At times, however, differentiating tumor from peritumoral edema is just as difficult with MRI as it is with other modalities, such as CT. Delineating tumor from healthy tissue is crucial for management and prognosis. In the case of osteogenic sarcoma, for example, MRI not only helps physicians plan an approach for biopsy and surgical procedures, it also helps them plan whether surgical intervention will consist of a limb-salvage procedure, an amputation, or a disarticulation.

Another important contribution of MRI is the monitoring of a tumor's response to adjuvant therapy—both preoperatively and postoperatively. Tumors responding to radiation or chemotherapy generally become more sharply marginated and decrease in size and vascularity.

Magnetic resonance imaging effectively helps characterize, stage, and follow musculoskeletal tumors and now is accepted as the imaging study of choice for such tumors.

Robert D. Boutin, M.D.
Michael R. Williamson, M.D.
Albuquerque, New Mexico

REFERENCES

Dalinka MK, Zlatkin MB, Chao P, Kricun ME, Kressel HY: The use of magnetic resonance imaging in the evaluation of bone and soft-tissue tumors. *Radiol Clin North Am* 1990; 28:461-70.

Gold Rl, Seeger LL, Bassett LW, Steckel RJ: An integrated approach to the evaluation of metastatic bone disease. *Radiol Clin North Am* 1990; 28:471-83.

Manaster BJ, Ensign MF: Imaging of musculoskeletal tumors. *Semin Oncol* 1991; 18:140-9.

Murphy WA: Imaging bone tumors in the 1990s. *Cancer* 1991; 67:1169-76.

Seeger LL, Widoff BE, Bassett LW, Rosen G, Eckardt JJ: Preoperative evaluation of osteosarcoma: Value of gadopentetate dimeglumine-enhanced MR imaging. *AJR* 1991; 157:347-51.

Vena Cava Filters

WHILE most patients with deep vein thrombosis can be successfully treated with anticoagulation therapy alone, some require the placement of an inferior vena cava filter, either as an alternative form of treatment or as adjunctive treatment along with anticoagulation, for prevention of pulmonary embolism. Generally accepted indications for placement of a vena cava filter include: absolute contraindication to anticoagulation; a complication of anticoagulation (such as hemorrhage); a relative contraindication to anticoagulation, such as patients prone to falling; the presence of a large amount of free-floating clot; short-term contraindications to anticoagulation, such as brain biopsy; deep vein thrombosis with unsuspected pulmonary embolism diagnosed at presentation; and a massive pulmonary embolism with lower extremity deep vein thrombosis. Since placement of a vena cava filter does not treat the underlying deep vein thrombosis and anticoagulation alone does not protect against pulmonary embolism in patients with a large volume of free-floating clot, both anticoagulation therapy and vena cava filter placement are indicated in some patients.

The stainless steel Greenfield filter, in use for about 15 years, has traditionally been placed by surgical cutdown. Over the last several years, percutaneous placement by interventional radiologists has been increasing in popularity. Also, four other vena cava filters are now available. All are designed for percutaneous placement under local anesthesia without a surgical cutdown. They are placed most often via the right common femoral vein, but not uncommonly via the right internal jugular vein. Alternate sites include the left common femoral vein and, for the Simon nitinol filter, the antecubital vein. The location of the renal veins and the diameter of the inferior vena cava are determined by venography before filter insertion.

The ideal vena cava filter should be a device that is easily and safely placed percutaneously, biocompatible, mechanically stable, and able to trap threatening emboli while not causing occlusion of the vena cava. With the stainless steel Greenfield filter, the rates of vena cava occlusion and of recurrent pulmonary embolism appear to be about 5%. When placed properly, this filter very rarely migrates. The outer diameter of the introducer sheath is 29.5 F, so the vein at the entry site must be dilated to about 10 mm to accommodate the introducer. The filter is ferromagnetic and can cause artifacts on magnetic resonance images, totally obscuring or distorting the appearance of adjacent structures in the spine, retroperitoneum, or abdomen.

Several vena cava filters recently have undergone clinical trials in the United States and Europe. Most of the efforts in the design of these new filters have been directed towards producing an effective filter that can be placed through a smaller introducer. Although accurate data in large numbers of patients are not available, the vena cava patency rate and recurrent pulmonary embolism rate for these new filters appear to be similar to those reported for the stainless steel Greenfield filter. The titanium Greenfield filter, Vena Tech filter, and Simon nitinol filter are nonferromagnetic, causing little or no artifacts on magnetic resonance images. An advantage of the bird's nest filter is that it can be placed into large venae cava, up to 40 mm in diameter. The Simon nitinol filter is delivered with a relatively flexible 7 F catheter, so it can be placed via an antecubital vein.

While there is probably no single "best" vena cava filter, many options are now available to the interventional radiologist. Percutaneous placement of inferior vena cava filters when done by physicians skilled in fluoroscopically monitored catheter and guide wire techniques, including performance and interpretation of vena cavagrams, is a relatively safe procedure. These filters clearly have a role in preventing pulmonary embolism.

Jerry N. King, M.D.
Anna M. Champlin, M.D.
Robert N. Ashby, M.D.
Albuquerque, New Mexico

REFERENCES

Dorfman GS: Percutaneous inferior vena caval filters. *Radiology* 1990; 174:987-92.

Dorfman GS, Cronan JJ, Paolella LP, et al: Iatrogenic changes at the venotomy site after percutaneous placement of the Greenfield filter. *Radiology* 1989; 173:159-62.

Kim D, Schlam BW, Porter DH, Simon M: Insertion of the Simon nitinol caval filter: Value of the antecubital vein approach. *Am J Roentgenol* 1991;157:521-2.

Pelvic Magnetic Resonance Imaging

ULTRASONOGRAPHY is the established screening method for evaluating the pelvis and is often the only radiographic study needed for patients with pelvic disease. As techniques continue to be refined, however, there are now several clinical indications when magnetic resonance imaging (MRI) may give more information.

Magnetic resonance imaging has been shown to be superior to both ultrasonography and computed tomography in the staging of tumors arising from the endometrium or cervix. Recent studies have shown that administering gadolinium-labeled pentetate frequently may aid in evaluating the extent of spread of endometrial tumors and, occasionally, in the staging of more advanced cervical lesions.

Leiomyomas have a characteristic appearance on MRI, and although ultrasound can diagnose them easily, when infertility patients are to undergo myomectomy procedures, MRI can better define the number and location of leiomyomas. This enables the surgeon to have a detailed preoperative map of the lesions present. Magnetic resonance can also be used in the follow-up of patients who are receiving therapy with gonadotropin analogues for leiomyoma shrinkage.

When women present with a pelvic mass during pregnancy, MRI can be very useful in defining the nature and extent of the mass, thereby differentiating uterine leiomyomas (which can be observed during pregnancy) from adnexal masses (which may have to be removed).

Magnetic resonance imaging now is used more frequently in evaluating all types of congenital anomalies of the pelvis. Although its role in evaluating uterine (Mullerian duct) anomalies has been established for some time, it can also be used to define the abnormal anatomy present in infants with ambiguous genitalia. In neonates with anal atresia, MRI can define accurately the level of the obstruction, thereby helping to determine the type of surgical procedure that should be done and, at the same time, indicate possible associated anomalies of the kidneys and spine.

Rosalind B. Dietrich, M.D.
Orange, California

REFERENCES

Gambino J, Caldwell B, Dietrich RB, Walot I, Kangarloo H: Congenital disorders of sexual differentiation Findings on MR imaging. *AJR* 1992, in press.

Hricak H, Hamm B, Semelka R, et al: Carcinoma of the uterus: Use of gadopentetate dimeglumine in MR imaging. *Radiology* 1991; 181:95-106.

Kier R, McCarthy S, Scoutt LM, Viscarello RR, Schwartz PE: Pelvic masses in pregnancy: MR imaging. *Radiology* 1990; 176:709-13.

Magnetic Resonance Angiography in the Head and Neck

MAGNETIC resonance angiography (MRA) is an exciting application of magnetic resonance imaging. This technique creates a large number of thin, contiguous slices that can be viewed individually or reconstructed into a slab of images that are collapsed into a three-dimensional projection. These collapsed images allow the visualization of entire vessels. A cine loop may be obtained from a series of rotated MRA images in order to evaluate vascular anatomy rapidly. Choosing the best imaging parameters and the plane of imaging determines the success rate of this procedure.

Magnetic resonance angiography is a useful tool for evaluating patients with cerebral ischemia and stroke and for demonstrating occlusive disease in the cervicocranial, intracranial carotid, and vertebrobasilar circulations. Nearly all arterial stenoses can be shown well, although typically they are overgraded. Magnetic resonance angiography examinations can be used to show the whole length of the carotid and vertebral vessels from their origins in the neck to their intracranial branches. Occlusive lesions of the intracranial arteries may also be identified, provided the vessels are of sufficient caliber and the intracranial vasculature is depicted.

Magnetic resonance angiography is ideal for placing an arterial occlusive lesion into one of three diagnostic groups—normal; minor stenosis of a vessel not impeding flow; or severe flow-limiting stenosis. These categories will indicate whether or not a patient needs to undergo conventional angiography or whether medical treatment is indicated.

Carotid ulcerations may not be detected with MRA because the relative stasis of blood within these lesions fails to produce adequate blood flow contrast. In such cases, duplex sonography of the carotid bifurcation can be useful. On the other hand, MRA can show the whole length of the internal carotid artery, which sonography is generally unable to do because of the mandible and skull base. Another pitfall of MRA relates to the overgrading of vascular stenoses. This error is produced by the presence of turbulent flow distal to the stenoses, which results in signal loss caused by dephasing.

The current application of MRA in intracranial aneurysms is limited to certain clinical situations such as evaluating patients with severe pulsating headaches or ocular migraine headaches. Magnetic resonance angiography can be used as a noninvasive screening examination in patients with a family history of or a phobia about aneurysms. Patients with subarachnoid hemorrhage must undergo a conventional angiogram. Magnetic resonance angiography has proven to be a good method of detecting and characterizing intracranial arteriovenous malformations. A variety of techniques have been used to delineate the nidus and major feeding vessels of these malformations.

Displacement or compression of vessels is another use of MRA in evaluating patients with intracranial tumors. The direction of displacement of a vessel can aid in deciding the proper surgical approach and in anticipating the structures as they are surgically identified. A neoplasm encasing a vascular structure can also be clearly depicted. Such encasement may involve either an arterial structure or the cortical veins and dural sinuses.

Magnetic resonance angiography is a useful technique for providing information about the intra- and extracranial carotid and vertebrobasilar circulations. When used in conjunction with the history and clinical findings, it can help physicians diagnose and treat patients with occlusive vascular diseases. Magnetic resonance angiography cannot substitute for conventional angiography in all cases and is not generally indicated in patients with subarachnoid hemorrhage.

Anton N. Hasso, M.D.
Loma Linda, California

REFERENCES

Caplan LR, Wolpert SM: Angiography in patients with occlusive cerebrovascular disease. Views of a stroke neurologist and neuroradiologist. *AJNR* 1991; 12:593-601.

Edelman RR, Mattle HP, Atkinson DJ, Hoogewoud HM: MR angiography. *AJR* 1990; 154:937-46.

Masaryk AM, Ross JS, DiCello MC, Modic MT, Paranandi L, Masaryk TJ: 3DFT MR angiography of the carotid bifurcation: Potential and limitations as a screening examination. *Radiology* 1991; 179:797-804.

Ross JS. Masaryk TJ, Modic MT, Ruggieri PM, Haacke EM, Selman WR: Intracranial aneurysms: Evaluation by MR angiography. *AJR* 1990; 155:159-65.

Intraoperative Radiotherapy

IN clinical radiotherapy, higher doses of radiation increase the probability of tumor cure at the expense of normal tissue. Intraoperative radiotherapy offers a means to irradiate the tumor selectively, thus improving the therapeutic ratio. Intraoperative radiotherapy allows more precise localization of the target volume under direct vision while protecting normal tissue by manual displacement or shielding. Intraoperative radiotherapy also takes advantage of the increased biologic effect of a large single dose of radiation.

Typically, following diagnosis and staging by a multidisciplinary team, patients receive a moderate dose preoperatively of external beam irradiation (45 to 50 gray for 4.5 weeks to 5 weeks) to a relatively large area, including the tumor and regions of suspected subclinical extension. After a break of about four weeks, the tumor is resected, if possible, and intestinal or biliary bypasses are done, if needed. At this point, the surgeon and radiation oncologist jointly determine the areas at greatest risk for recurrence, and those areas receive a single large fraction (10 gray to 20 gray) of intraoperative radiotherapy. The patient can be treated under anesthetic either in the operating room or the radiation therapy department.

The most promising uses for intraoperative radiotherapy are in gastric and rectal cancer. Data from studies in Japan indicate improvement in survival rates following surgical procedures and intraoperative radiotherapy in gastric tumors, compared with resection alone. The benefit is greatest in higher stage tumors. Data from this country show improved local control and survival in initially unresectable rectal carcinomas when intraoperative radiotherapy is added to preoperative irradiation and resection. Other sites where this therapy has shown promise include retroperitoneal sarcomas, mediastinal tumors, and biliary tumors. Toxicity has been minimal, and extensive data from studies in large animals detail the sensitivities of a wide range of tissues and organs to single large radiation fractions.

Intraoperative radiotherapy offers the possibility of improved local tumor control, which may increase the probability of tumor cure.

Henry Z. Montes, M.D.
David S. Shimm, M.D.
Tucson, Arizona

REFERENCES

Hoekstra H, Sindelar W, Kinsella T: Surgery with intraoperative radiotherapy for sarcomas of the pelvic girdle: A pilot experience. *Int J Radiat Oncol Biol Phys* 1988; 15:1013-6.

Kinsella T, DeLuca A, Barnes M, Anderson W, Terrill R, Sindelar W: Threshold dose for peripheral neuropathy following intraoperative radiotherapy in a large animal model. *Int J Radiat Oncol Biol Phys* 1991; 20:697-701.

Infections in Emergency Medicine—Newer Imaging Strategies

INFECTIONS are a common problem in emergency medicine. Newer imaging methods may expand the role of emergency radiology in the preliminary evaluation of some infectious and inflammatory processes.

Acute sinusitis can be evaluated with a limited sinus computed tomographic (CT) scan using 5 mm axial sections without contrast—one through the frontal sinuses, a second through the sphenoid-ethmoid level, and two through the maxillary sinuses. Sinus opacification, air-fluid levels, osseous changes, and polypoid lesions are easily seen in under ten minutes. If more serious conditions are suggested, the patient can have an elective complete CT sinus study, but limited CT is more useful and no more costly than plain films.

Normal appendices are found in 15% to 25% of appendectomies for suspected acute appendicitis. Although deaths are rare, the morbidity and cost of negative laparotomy are substantial. Those in whom improved preoperative diagnosis is of particular value include women of reproductive age, where the normal appendix rate is 35% to 45%; pregnant patients where abortion is a consideration; very young, older, immunosuppressed, and other patients with atypical presentations.

Graded compression sonography, combined with transabdominal or transvaginal ultrasound, can visualize an abnormal appendix directly and show an alternative pathologic process, particularly gynecologic disease. In this study, the operator uses the transducer to gradually compress the abdomen at the site of pain, while searching for a dilated, thick-walled appendix, abnormal fluid collection, or appendicolith. Accuracies of 80% to 90% have been reported, but the method is operator dependent, and sensitivity is reduced if the appendix has perforated. Computed tomography may also show an abnormal appendix and is particularly useful in the 5% of patients who present with right lower quadrant mass, often distinguishing between phlegmon and abscess and helping with percutaneous drainage of the latter.

Patients with acute osteomyelitis usually do not have findings on plain radiographs seven to ten days after the onset of symptoms. Nuclear medicine technetium 99m (^{99m}Tc)-MDP bone scans are 95% sensitive but are not specific. A negative study result 24 hours after the onset of symptoms, however, essentially rules out acute bone infection. The three-phase bone scan improves specificity, often differentiating osteomyelitis from cellulitis, a common problem in the feet of diabetic patients. Specificity is further increased by the use of indium 111-labeled oxine or ^{99m}Tc-HMPAO leukocyte scans in conjunction with bone scans, but these scans take 24 hours to complete.

Magnetic resonance imaging (MRI) is as sensitive as and more specific than ^{99m}Tc-labeled medronate disodium bone scintigraphy, showing marrow as well as soft-tissue and periosteal changes. It can be done in less than two hours, but it is costly, not universally available, and, like bone scans can occasionally result in false-positive results, particularly in the setting of trauma, previous surgical procedures, or neuropathic joint disease . In suspected vertebral osteomyelitis, however, MRI is the method of choice. Multiplanar imaging capabilities facilitate the depiction by MRI of vertebral body marrow alterations, disc involvement, and paraspinal or epidural abscess with unsurpassed clarity.

Michael I. Zucker, M.D.
Lawrence Yao, M.D.
Los Angeles, California

REFERENCES

Balthazar EJ, Megibow AJ, Siegel SE, Birnbaum BA: Appendicitis: Prospective evaluation with high-resolution CT. *Radiology* 1991; 180:21-4.

Brown JJ: Acute appendicitis: The radiologist's role. *Radiology* 1991; 180:13-14.

Gold RH, Hawkins RA, Katz RD: Bacterial osteomyelitis: Findings on plain radiography, CT, MR, and scintigraphy. *AJR* 1991; 157:365-70.

Larson JM , Peirce JC, Ellinger DM , et al : The validity and utility of sonography in the diagnosis of appendicitis in the community setting. *AIR* 1989; 153:687-91.

Picture Archiving and Communication Systems

WITH the introduction of computed tomography (CT) and magnetic resonance imaging (MRI), an ever-increasing proportion of procedures done in radiology are acquired in a digital format. In addition, computed radiography (CR) which uses phosphostimulable plates instead of film, may replace conventional radiography, especially for portable examinations of the chest.

Picture archiving and communication systems (PACS) acquire, transmit, store, and display image information. Besides acquiring images directly from digital modalities (CT, MRI, and nuclear medicine) and from CR, these systems use laser digitizers to convert images on radiographic film to digital images. The images stored in PACS can be reproduced either on film with a laser camera or displayed using a high-resolution television monitor. Picture archiving and communication systems are expected eventually to replace most radiographic film. Reducing the operating costs associated with handling and storing radiographs and eliminating film and film processing may make these systems cost-effective in spite of the initial large expenditures for equipment.

When images are in digital form, they can be manipulated or processed by using window and level controls (intensity and contrast), inverse intensity (white on black to black on white), or edge-enhancement (unsharp masking). For a particular patient, images from the various modalities or from previous studies can be placed on the same or associated monitors for comparison. Such systems obviously would speed the retrieval and display of images. Treatment can be initiated earlier for patients in an Intensive Care Unit when digital images are readily available. The frequency of communication between the radiologist and the referring physician may decline, however, because in some instances the images are being viewed before the radiologist's interpretation is received.

The spatial and contrast resolution of the current digital modalities can be faithfully reproduced with PACS, especially if film is used for the primary interpretation. Although most investigators would agree that soft-copy display is also more than adequate, there still are concerns about the contrast gray levels that can be seen without varying the window and level on these displays. The simultaneous display of a large number of images is difficult. Also, subtle bone abnormalities, microcalcifications in mammography, and perhaps pneumothoraces are still somewhat difficult to see on these displays.

To date, only a handful of large institutions have successfully implemented clinical PACS. The reasons for the slow introduction of this technology are multiple, but two issues are clearly paramount: capital expense and resolution limitations. In one study examining the cost of PACS, the potential for substantial reductions in operating costs to offset capital depreciation was shown. This study, however, did not include a commercial manufacturer's cost for research and development, liability insurance, or a profit margin. Almost all of the savings that can be realized with PACS are related to the current digital modalities with little, if any, savings to be accrued on the general radiographic side (CR or digitizing radiographs).

In addition to the possible reduction in operating costs and improved efficiency in the handling of medical images, PACS could enhance medical education by providing sophisticated retrieval capability from the image database based on radiologic or pathologic findings.

Ronald L. Arenson, M.D.
San Francisco, California

REFERENCES

Arenson RL, Seshadri SB, Kundel HL, et al: Clinical evaluation of a medical image management system for chest images. *AJR* 1988; 150:55-9.

DeSimone DN, Kundel HL, Arenson RL, et al: Effect of a digital imaging network on physician behavior in an intensive care unit. *Radiology* 1988; 69:41-4.

Shoulder Magnetic Resonance Imaging

THE strength and stability of the most mobile but least stable joint in the body, the glenohumeral joint, is provided by the four muscles of the rotator cuff. The supraspinatus, infraspinatus, teres minor, and subscapularis muscles form a musculotendinous cuff that envelopes the humeral head and glenohumeral joint. The supraspinatus muscle is interposed between the humeral head and coracoacromial arch.

The impingement syndrome is a common cause of shoulder pain and may cause 95% of rotator cuff tears. When the soft tissue structures of the rotator cuff become entrapped between the coracoacromial arch and the greater tuberosity of the proximal humerus during abduction and elevation of the arm, a series of pathologic events may occur. Subacromial bursitis and rotator cuff tendinitis occur first with progression to fibrosis and rupture of the rotator cuff later. The impingement may occur at the site of the anterior acromion at the acromioclavicular joint due to spurring, or along the undersurface of the coracoacromial ligament.

Most rotator cuff tears affect the supraspinatus tendon 1 cm proximal to its site of insertion, the "critical zone," near its attachment to the humeral tuberosity. Anterior and posterior extensions may occur, but less frequently. Complete tears are defined as full thickness tears of the rotator cuff. Partial tears are incomplete tears that do not allow fluid to flow between the glenohumeral joint and the subacromial bursa.

The signs and symptoms of rotator cuff tears are not specific. Consequently, the diagnosis is often delayed until a complete tear has occurred. The goal of evaluating shoulder pain should be to determine the cause before irreversible tendon damage has occurred.

Magnetic resonance imaging (MRI) provides excellent depiction of the anatomic structures of the shoulder, including the rotator cuff, the long head of the biceps tendon, articular capsule, muscles, and bone. The improved contrast and additional imaging planes of MRI provide the orthopedic surgeon with anatomic information regarding the presence of tendinitis versus a partial tear versus a complete tear of the rotator cuff. This information, in addition to the clinical presentation, assists the surgeon in determining treatment, that is, physical therapy, arthroscopy, or an open surgical procedure.

The MRI findings of tendinitis are thickening of the cuff tendon and intermediate (grey) signal replacing the normal low signal (black) tendon. Partial tears show a focal thinning of the tendon and may have fluid within the niche. With complete rotator cuff tears, the normal low-intensity signal of the tendons is interrupted by a bright signal indicating discontinuity of the rotator cuff. The (high signal) discontinuity may be filled by either fluid, granulation tissue, or hypertrophied synovium.

Compared with arthroscopy, MRI showed a sensitivity of 100% and specificity of 88% in evaluating rotator cuff tears and a sensitivity of 92% and a specificity of 100% compared to a surgical procedure. Magnetic resonance imaging is able to show bony impingement on the rotator cuff tendon and can indicate other causes of shoulder pain, including effusions, occult fractures, metastases, avascular necrosis, labral tears, and loose bodies. Magnetic resonance gadolinium arthrography may be indicated in cases of dynamic instability, failed surgical procedures for rotator cuff tears, negative noncontrast MRI, and in differentiating partial tears from tendinitis.

Joel K. Lipman, M.D.
Newport Beach, California

REFERENCES

Burk DL Jr, Karasick D, Kurtz AB, et al: Rotator cuff tears: Prospective comparison of MR imaging with arthrography, sonography, and surgery. *AJR* 1989; 153:87-92.

Flannigan B, Kursunoglu-Brahme S, Snyder S, Karzel R, Del Pizzo W, Resnick D: MRI arthrography of the shoulder: Comparison with conventional MR imaging. *AJR* 1990; 155:829-32.

Kieft G, Bloem J, Rozing P, Obermann W: Rotator cuff impingement syndrome: MR imaging. *Radiology* 1988; 166:211-4.

The Imaging of Impotence

IMPOTENCE affects more than 10 million men in the United States. Clinical evaluation usually can exclude endocrinologic imbalance, neurogenic dysfunction, and psychologic problems as etiology. A patient who fails to get an erection after vasoactive medications are injected probably has hemodynamic impotence. Dynamic studies that include imaging techniques are now available to discriminate between arterial and venous pathology.

Doppler ultrasound with color flow and spectral analysis, dynamic infusion corpus cavernosometry and cavernosography, and selective internal pudendal arteriography are outpatient diagnostic procedures that will differentiate, image, and quantify the abnormalities in patients with hemodynamic impotence. Not all tests are needed in every patient. Each of these examinations is preceded with the intracavernosal injection of vasoactive medication. Papaverine hydrochloride, phentolamine mesylate, or prostaglandin E_1 will overcome normal sympathetic tone and produce an erection by smooth muscle relaxation and arterial dilatation in a normal patient.

Color-flow Doppler and spectral analysis will show the cavernosal arteries and can identify the hemodynamic effects of stricture or occlusion. Peak systolic velocity is measured. Normal ranges are well established. Spectral analysis also is used to predict the presence of venous disease. Sizable venous leaks in the dorsal penile vein are readily imaged. While the technique may not adequately identify low-grade venous pathology, it will identify the size and location of fibrous plaque formation associated with Peyronie's disease.

Cavernosography or cavernosometry is a separate procedure that will quantitate the severity of venous incompetence as well as specifically identify the various avenues of systemic venous return that must be localized if venous occlusive therapy is chosen. In this study, the peak arterial systolic occlusion pressure is quantified during erection, and the presence of arterial pathology can be confirmed. The arterial data are not as reliable as the ultrasound-obtained data because they rely on audible Doppler, which can be obscured in the underlying "noise" heard with erection. The arterial data obtained with both of these examinations are quantitative and replace the qualitative audible Doppler used previously. Specialized equipment (Life Tech, Houston, Texas) allows dynamic data acquisition, ensuring that the needed information is obtained at peak stimulation.

Arteriography is done only if reconstructive surgery is contemplated. The examination includes subselective catheterization of the internal pudendal arteries, magnification technique, and evaluation of the recurrent epigastric arteries, which will be harvested for revascularization. An arterial operation is usually successful in younger patients with perineal trauma with a single point of stenosis or occlusion but has been least successful in those patients with atherosclerosis because of the multifocal nature of this disease.

Diagnostic information available today is more specific and reliable than at any time in the past and can realistically estimate the severity of the hemodynamic disorder and allow individual treatment options. Ongoing studies will show whether the data these studies provide make an important difference in the treatment of vascular impotence.

Roger H. Thomas, M.D.
Newport Beach, California

REFERENCES

Goldstein I, Greenfield A: Pathophysiology and physiology of impotence, *In* Impotence and Prostate Disease—Imaging and Intervention: The Team Approach (syllabus). Minneapolis, University of Minnesota, 1989.

Krysiewiczk S, Mellinger BC: The role of imaging in the diagnostic evaluation of impotence. *AJR* 1989; 153:1133-9.

Mueller S, von Wallenberg-Pachaly H, Voges G, Schild HH: Comparison of selective internal iliac pharmaco-angiography, penile brachial index, and duplex sonography with pulsed Doppler analysis for the evaluation of vasculogenic (arteriogenic) impotence. *J Urol* 1990: 143:928-32.

Thomas R: Imaging determines cause of hemodynamic impotence. *Diagn Imaging* 1990: 12:102-7.

Current Status of Discography

DISCOGRAPHY is a physiologic evaluation of intervertebral discs consisting of volumetric, manometric, pain provocative, and radiographic appearance. Since its introduction in 1948, discography has gained and lost popularity several times. It suffered a major setback in 1968, after reports of 37% false-positive results in normal volunteers. The literature is still replete with articles that have reported its many advantages and applications and have considered it an essential guide to planning operations on the spine.

The procedure consists of injecting contrast material under fluoroscopic control via an extradural or intradural approach. The injection in a normal disc is painless—the patient feels only a pressure sensation—and the contrast agent remains in the nucleus pulposus. The test result typically is considered positive when injection creates pain that simulates the patient's symptoms and is associated with an abnormal morphologic discogram appearance or contrast extravasation, or both. An 89% parallel correlation of magnetic resonance imaging and discography has been reported.

Discography is indicated when magnetic resonance imaging, computed tomography, or a myelogram shows negative or equivocal findings for disc disease; when there is a positive result on these tests indicating disc disease at multiple levels—in this case, discograms are used to determine the symptomatic level or levels; in the case of recurrent low back pain after a surgical procedure to distinguish scar tissue from recurrent disc herniation; to evaluate the discs above or below the proposed spinal fusion; and as a test for an injection of cortisone.

Although discography is a relatively safe procedure, a discitis rate of 1% to 4% has been reported. Allergic reactions to nonionic contrast agents are uncommon, and epidural abscess is extremely rare. The total cost for a three-disc-level procedure is currently between $1,000 and $2,000, including professional and hospital fees. Despite the clouds of controversy surrounding discography, it remains the only provocative test available and is indicated as a complementary test before a surgical procedure is done on the spine.

Jamshid Tehranzadeh, M.D.
Orange, California

REFERENCES

Bernard TN Jr: Lumbar discography followed by computed tomography. Refining the diagnosis of low-back pain. *Spine* 1990; 15:690-707.

Colhoun E, McCall IW, Williams L, Cassar Pullicino VN: Provocation discography as a guide to planning operations on the spine. *J Bone Joint Surg* 1988; 70(B):267-71.

Osti OL, Fraser RD, Vernon-Roberts B: Discitis after discography. The role of prophylactic antibodies. *J Bone Joint Surg* 1990; 72(B):271-4.

Radiosurgery for Tumors and Arteriovenous Malformations of the Brain

TUMORS and arteriovenous malformations located in deep, inaccessible areas of the brain are now treatable with radiosurgery, a technique that uses high doses of radiation to inactivate these lesions. Several different methods of radiosurgery have been developed, but all share the common characteristic of focusing multiple, precise, small radiation beams on an intracranial target. Although charged particle beams have been successfully developed for radiosurgery, the most widely available devices use photon radiation generated by a linear accelerator or a multiple cobalt 60 source machine ("Gamma Knife").

Radiosurgery is given in a single or a very small number of treatments, unlike conventional radiotherapy in which multiple fractions are delivered over a period of weeks. To do radiosurgery, a stereotactic localization device is fixed to the patient's skull; this apparatus provides a coordinate system for defining the position of the lesion and its relationship to surrounding brain structures. Computed tomography scans are done to determine the three-dimensional configuration of the lesion; angiography and magnetic resonance scans may also be useful. Computed radiation treatment planning is used to design radiation dose distributions that conform to the shape of the target, confining the areas of high dosage to the lesion, with less irradiation of surrounding brain tissue. Radiosurgery is done—usually on an outpatient basis—with the patient's head immobilized in the localization device.

Excellent results have been obtained using radiosurgery for patients with inoperable arteriovenous malformations. Most treated with radiosurgery—60% to 90%—are completely obliterated as documented by follow-up angiography. The risk of cerebral hemorrhage is eliminated provided the malformation is totally obliterated. Regression of the lesion is slow after radiosurgery, however, taking one to three years to complete. The risk of bleeding persists during the interval between treatment and complete obliteration. Radiosurgery can be used as the sole modality to treat small arteriovenous malformations—less than 3 cm—or can be combined with angiographic embolization for more extensive lesions.

A variety of benign and malignant brain tumors have been treated with radiosurgery, including acoustic neuromas, meningiomas, pineal and pituitary region tumors, gliomas, posterior fossa neoplasms and metastatic lesions. For selected small, noninvasive tumors, radiosurgery alone may be used, while for larger neoplasms, surgical debulking can be initially used, followed by radiosurgery to treat the residual tumor. Radiosurgery is particularly useful for tumors that have recurred after a surgical procedure and conventional radiation therapy, provided the volume of recurrent disease is small.

Brian R. Griffin, M.D.
Seattle, Washington

REFERENCES

Friedman WA, Bova BJ: Stereotactic radiosurgery. *Contemporary Neurosurgery* 1989; 11:334-42.

Larson DA, Gutin PH, Leibel SA, Phillips TL, Sneed PK, Wara WM: Stereotaxic irradiation of brain tumors. *Cancer* 1990; 65:792-9.

Loeffler JS, Alexander E 3d: The role of stereotactic radiosurgery in the management of intracranial tumors. *Oncology* 1990; 4:21-31.

Chemoembolization in the Treatment of Hepatic Malignancy

UNRESECTABLE hepatic malignancy, either primary or metastatic, carries a grave prognosis, with a usual life expectancy of three months to six months, and, unfortunately, the condition is common. Colon carcinoma, which frequently spreads to the liver, will take 60,000 American lives every year. Hepatocellular carcinoma (HCC), the most common fatal malignancy worldwide, is endemic in the Orient. With Pacific Rim migration, the incidence is expected to increase in the western United States. Response rates with systemic chemotherapy are about 30% and, in hepatic metastases caused by colon carcinoma, about 20%.

The poor prognosis and high prevalence have led to innovative treatments of liver tumors, including infusing chemotherapy directly into the hepatic artery (to increase the concentration delivered to the tumor) and blocking the blood supply with microscopic particles (to cause tumor ischemia). Chemoembolization is a combination of the two therapies in which chemotherapy and particles are infused simultaneously.

Three congruent circumstances permit the safe and effective application of such regional treatments: the liver has a dual blood supply with either the hepatic artery or portal vein capable of sustaining the organ; tumors of the liver are supplied by the hepatic artery; and the hepatic artery is nearly always accessible percutaneously with current catheterization techniques.

Patients eligible for chemoembolization include those in whom the tumor is unresectable yet confined to the liver. Initially, angiography of the celiac axis and superior mesenteric artery are done to determine the blood supply of the liver and status of the portal vein. A small (2F to 3F) catheter is placed coaxially through the larger (5F to 6F) guiding catheter past the intestinal and gallbladder vessels into the hepatic artery. The chemoembolization mixture is infused while monitored by fluoroscopy. Nausea and pain after the procedure are greatly relieved by the antiemetic ondansetron and intraarterial lidocaine, respectively. Complications such as embolization of the intestine, hepatic abscess, or infarction occur in less than 3% of patients. Response rates in hepatocellular carcinoma have ranged from 50% to 75%. In addition, most recent studies have involved patients who failed to respond to systemic chemotherapy.

Newer applications and broader use of hepatic chemoembolization are expected. Such advances may include the shrinkage of hepatic malignancies permitting curative resection, palliation until liver transplantation, safe use in patients with portal vein thrombosis, and the application in children with hepatoblastoma and hepatocellular carcinoma.

Michael J. Pentecost, M.D.
George P. Teitelbaum, M.D.
Los Angeles, California

REFERENCES

Mavligit GM, Charnsangavej C, Carrasco CH, Patt YZ, Benjamin RS, Wallace S: Regression of ocular melanoma metastatic to the liver after hepatic arterial chemoembolization with cisplatin and polyvinyl sponge. *JAMA* 1988; 260:974-6.

Venook AP, Stagg RJ, Lewis BJ, et al: Chemoembolization for hepatocellular carcinoma. *J Clin Oncol* 1990; 8:1108-14.

Yamada R, Kishi K, Sonomura T, Tsuda M, Nomura S, Satoh M: Transcatheter arterial embolization in unresectable hepatocellular carcinoma. *Cardiovasc Intervent Radiol* 1990; 13:135-9.

Vasodilator and Exercise Cardiac Perfusion Scintigraphy

THE long anticipated Food and Drug Administration approval of two technetium 99m (^{99m}Tc)-based cardiac perfusion imaging agents and the coronary vasodilator dipyridamole for intravenous administration occurred early in 1991.

Dipyridamole increases coronary blood flow without exercise and improves the sensitivity of isotope imaging in those patients unable to exercise well. Side effects such as chest pain, headache, and dizziness are common but reversible with aminophylline.

The two ^{99m}Tc isonitrile agents differ greatly in biodistribution. The ^{99m}Tc-teboroxeme redistributes rapidly from myocardium. This has advantages for rapid sequential stress and rest imaging but demands rapid imaging following exercise. Ideally, the exercise is done in close proximity to a multi-head single photon emission computed tomography (SPECT) system.

The ^{99m}Tc-sestamibi agent remains fixed for hours in the myocardium proportional to regional blood flow at the time of injection. This has distinct logistic advantages. The physical distance of the laboratory from the nuclear medicine site or delays in completing examinations no longer compromise the study as they might with thallium 201 (^{201}T1). The ^{99m}Tc not only reflects perfusion but also shows myocardial viability better than ^{201}T1. Areas of ischemia appearing as infarcts are less common.

Both ^{99m}Tc compounds have distinct physical advantages over ^{201}T1. The 140 kiloelectron volt (keV) photons are less attenuated by tissue and better suited for SPECT imaging than ^{201}T1 (80 keV). The shorter half-life—six hours versus 73 hours—allows more activity with less radiation absorbed. Initial reports indicate comparable or favorable results compared with ^{201}T1, and further experience is likely to show improved results as the physical advantages are exploited by changing techniques. Cost is comparable and may even be less per study in high-volume departments.

Positron studies have distinct physical advantages over all SPECT agents. With very high-energy (511 keV), short-lived isotopes, attenuation by tissue is minimal and specificity is higher than SPECT. Combined perfusion and glucose metabolism are the gold standard for noninvasive assessment of viability. The high cost and limited availability are major disadvantages.

As the physical advantages of the ^{99m}Tc compounds are exploited to improve the specificity of SPECT imaging, they will have an important effect on the evaluation of coronary disease by scintigraphy. Vasodilator infusion has already improved scintigraphy results in those unable to exercise well.

Robert W. Henderson, M.D.
Pasadena, California

REFERENCES

Beller GA: Pharmacologic stress imaging. *JAMA* 1991; 265:633-8.

Liu P: Technetium-99m-sestamibi: Another window on myocardial viability? *J Nucl Med* 1991; 32:298-9.

Wackers FJ, Berman DS, Maddahi J, et al: Technetium-99m hexakis 2-methoxyisobutyl isonitrile: Human biodistribution, dosimetry, safety and preliminary comparison to thallium-201 for myocardial perfusion imaging. *J Nucl Med* 1989; 30:301-311.

The Simplicity and Safety of Radiologically Placed Gastric Tubes

ENDOSCOPISTS and, more recently, radiologists have shown that successful gastric intubation, once strictly a surgical procedure, can be done outside the operating room without an incision.

The procedure is done with a fluoroscope while the patient is fully awake. The stomach is inflated with air via a nasogastric tube, which pushes the transverse colon away from the abdominal wall. An appropriate site on the skin surface is prepared and then anesthetized with local anesthesia. For patients in whom a nasogastric tube cannot be passed, the stomach can be inflated through a skinny needle placed percutaneously into the stomach bubble. Under fluoroscopic guidance, a needle is inserted into the gastric lumen through which a guide wire is placed. The needle is removed and the tract is dilated to the desired width (usually 12 F to 16 F). A feeding tube, which usually has some form of anchor, such as a self-retaining pigtail loop, is then inserted. Some prefer to secure the stomach to the abdominal wall using small T-anchors introduced through separate needle punctures, but we have found that unnecessary.

An advantage of radiologic guidance is that ultrasound can be used to locate the spleen or liver should they be near the proposed entry site, and the colon can be quickly filled with air or contrast material if it is difficult to see with the fluoroscope. If or when a jejunal tube is needed, then the G-tube is readily replaced with a longer tube that is fluoroscopically guided into the small bowel. After bowel sounds return, usually within 24 hours, the tube is then ready to be used for feedings. The procedure usually takes under 30 minutes to do, and, if the tube should stop working after the tract matures—usually by two to four weeks—it is easily exchanged in a few minutes over a guide wire on an outpatient basis.

Because of the distended stomach, air can escape into the peritoneal cavity, which is rarely, if ever, of any clinical consequence. Morbidity and mortality data show that fluoroscopically directed feeding tubes can be placed with the same, and perhaps fewer, complications than surgically or endoscopically placed tubes. That, with the added benefits of no anesthesia or operating room charges, makes the radiologically placed feeding tube an attractive alternative for patients in need of enteral nutrition.

Jeffrey C. Brandon, M.D.
Larry-Stuart Deutsch, M.D., C.M.
Lane Kannegieter, M.D.
Damon P. Miller III, M.D.
Orange, California

REFERENCES

Halkier BK, Ho CS, Yee AC: Percutaneous feeding gastrostomy with the Seldinger technique: Review of 252 patients. *Radiology* 1989; 171:359-62.

Hicks ME, Surratt RS, Picus D, Marx MV, Lang EV: Fluoroscopically guided percutaneous gastrostomy and gastroenterostomy: Analysis of 158 consecutive cases. *AJR* 1990; 154:725-8.

O'Keeffe F, Carrasco CH, Charnsangavej C, Richli WR, Wallace S, Freedman RS: Percutaneous drainage and feeding gastrostomies in 100 patients. *Radiology* 1989; 172: 341-3.

ADVISORY PANEL TO THE SECTION ON RADIOLOGY
Charles A. Gooding, M.D.
Advisory Panel Chair
CMA Council on Scientific Affairs Representative
San Francisco

Richard S. Breiman, M.D.
CMA Section Chair
Oakland

Edward I. Miller, M.D.
CMA Section Secretary
Newport Beach

K. M. Tan, M.D.
CMA Section Assistant Secretary
Richmond

Anton N. Hasso, M.D.
Loma Linda University

Gary Glazer, M.D.
Stanford University

Richard Katzberg, M.D.
University of California, Davis

Richard M. Friedenberg, M.D.
University of California, Irvine

Hooshang Kangarloo, M.D.
Section Editor
University of California, Los Angeles

George Leopold, M.D.
University of California, San Diego

Ronald L. Arenson, M.D.
University of California, San Francisco

James Halls, M.D.
University of Southern California

Thomas G. Goergen, M.D.
California Radiological Society
Rancho Sante Fe

Jay C. Mall, M.D.
California Radiological Society
San Francisco

Paul S. Schulman, M.D.
California Radiological Society
Chula Vista

Christopher M. Rose, M.D.
California Radiation Oncology Association
Burbank

John To
Medical Student Representative
University of Southern California

50 Years Ago

From The Connecticut State Medical Journal

January 1943

The Beveridge Plan

REFERRED to by the noted British author Harold J. Laski as one of the vital documents of modern Britain, the recent report to the Parliament by Sir William Henry Beveridge on a plan for social security has already become significant to the English speaking world. The plan recommended for a system of compulsory State insurance presents a broad scheme to protect every man, woman and child in England from practically every personal insecurity and want. To the medical profession of the United States it does have important meaning, for that part of the plan devoted to medical care will be studied by all those in our country who are thinking of health problems and the public welfare.

It should be useful to consider in brief the main features of the Beveridge plan and these have been set down for us by Mr. Laski in a recent issue of the New York Times.

"First, it makes complete provision against unemployment. The wage-earner is freed from the haunting fear of insecurity.

"Second, it makes complete provision for a complete national health service for every man, woman and child.

"Third, it simplifies and unifies the whole system of workmen's compensation and introduces order into the present legal jungle in which insured workmen have lost themselves.

"Fourth, it protects the interest of the family by establishing endowment of all children after the first; it therefore relieves the pressure on husband and wife in a period where children, as the researches of Booth and Rowntree show, are the main causes of poverty, especially in the lowest income bracket.

"Fifth, it protects the aged and those, like the blind and deaf, who are at present hopeless dependents of charity.

"Sixth, it provides for the vital elements of life, childbirth, death and marriage, in each case modest-ly but far more adequately than at present.

"Seventh, it simplifies the whole process of administration by entrusting the objectives involved to a single Ministry of Social Security, where these are now entrusted to the overlapping zeal of some eight ministries.

"Eighth, it abolishes the necessity of the means test for relief—a method that has been one of the most fruitful causes of class hatred in the last generation.

"Ninth, it is a contributory scheme that on any rational calculation is well within the compass of the taxable capacity of the British nation.

"Tenth, it is simple, direct and intelligible—the main political administrative merits to be sought in any such scheme."

In spite of the "revolutionary" recommendations of the plan Mr. Laski believes it will be most difficult for the War Cabinet not to accept the plan generally and, if rejected, that it may not be possible for the Labor Party to continue in the National Government.

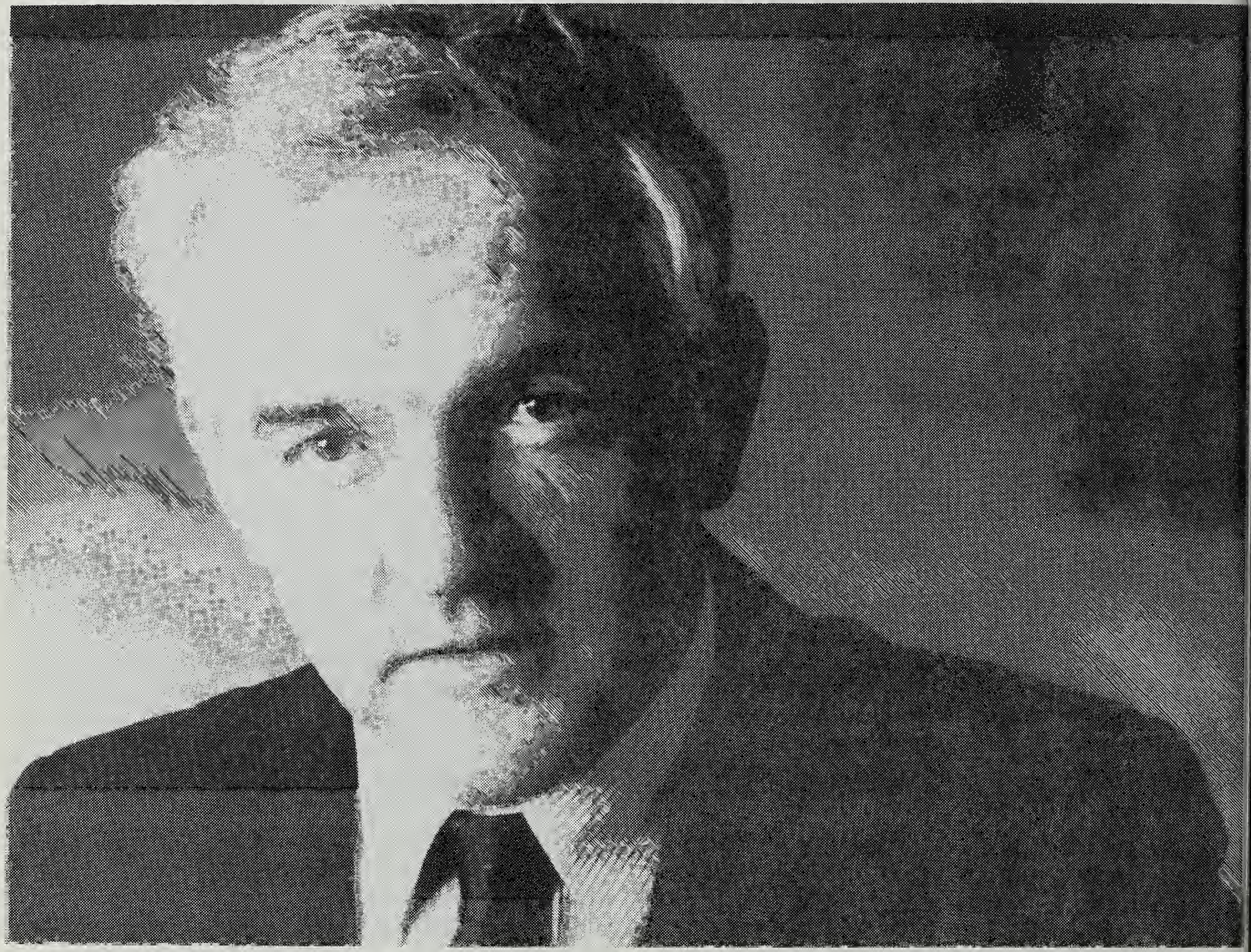
The primary interest for the medical profession of course, is that which pertains to health insurance. This will mean a national medical service in which the entire hospital system will be controlled by local and national authorities. On our own side of the Atlantic no such sweeping reforms have been seriously considered; nevertheless, most of the problems in Britain concerning health insurance are very like our own. The health of our industrial workers has already become one of our major problems and its solution may be eventually worked out along very broad lines. These recent words of Henry J. Kaiser before the National Association of Manufacturers may hold definite prophecy: "will the manufacturers now dare to organize, finance and manage medical centers in every industrial community, where medical service could be purchased on an insurance basis at a cost which would bring not only skill and facilities, but all the advantages of research within the reach of common man and at the same time provide the doctors of America with a participation which would adequately remunerate them for their long and expensive training?"

The physicians of America can no longer consider these and similar proposals merely as evolutionary trends, for they are unquestionably part of a definite movement toward the extension of medical practice on a broad social basis. It is only by being constantly aware of these things that the profession can hope to place itself in a position to aid in the direction of such thought to proper and practical channels for the common good.

Editorial from *The Connecticut State Medical Journal*, January 1943.

“Filling out insurance forms gives my patients a pain in the neck.”

—Dr. Patrick J. Carolan, Orthopedics, Bridgeport, CT



“Good medical care was once as easy as seeing your doctor. Complications set in. Forms to fill out. Deductibles. Hidden costs. Partial coverage. Non-medical intrusions into the patient-physician relationship.

“Doctors decided to do something about it. Through our State Medical Society, we first created **CSMS-IPA,** Connecticut’s only *statewide* IPA. Then we launched **M.D. Health Plan,** based on a simple principle: The best medicine for *patients* is practiced by *physicians*— not insurance companies, not benefit managers, not cost analysts.

“It worked! Now the fastest growing health plan in Connecticut and the choice of over 68,000 patients, our company is 100% owned and 100% directed by practicing physicians. Why not?”

Patrick Carolan, M.D., President, CSMS-IPA

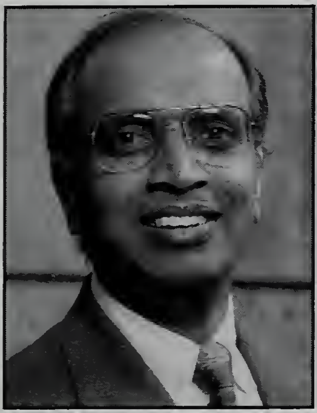
If *you* wish to join the 3,000 CSMS physicians who now participate, call us at one of the numbers below.

M.D. HEALTH PLAN

6 DEVINE STREET • NORTH HAVEN, CT 06473 • (203) 776-5759 • 1-800-345-9272

THE PRESIDENT'S PAGE

Cost of Health Care—The Truth



Will Rogers said: "If we ever pass out as a great nation we ought to put on our tombstone 'America died from a delusion that she had moral leadership.'" This moral leadership, which is lacking in our politicians including the legislative and executive branch, and is lacking in our corporate business structure, and, yes, even in our judiciary, has to be at least carried on by the people who take care of the sick. This moral leadership gives us authority to speak out the truth about the present health care mess we are in. Let me talk about the cost of health care, and the truth about health care costs. We all know that we spend over 12% of the GNP for our health care and we have about 33 million people who have either no insurance or poor insurance coverage. This leaves about 200 million with some good health care coverage.

Now let us talk about the reasons for the rising health care costs. First of all in the last decade or so our demographic and economic changes have been astounding. We have an aging population. Currently there are about 32 million Americans over the age of 65; 7.5 million of them need some form of long term care. About 1.5 million are in nursing homes. Those eighty-five years old and over represent the fastest growing group of our population. Our elderly account for 36% of our health care costs despite comprising about 12% of our population. Look at the economic changes. There are more people below the poverty line than a decade or two ago! Poor people consume more of the expensive therapeutic treatments rather than cheaper preventive measures. For example a person who goes without treatment for hypertension, ultimately ends up with stroke or heart disease which require costly treatments. Those who do not take care of their diabetes at an early stage end up in the emergency ward with ketoacidosis and consume costly services. Take a close look at other socioeconomic factors peculiar to our country. Every year more than 1.5 million Americans are victims of assault and over 2.5 million children are abused or neglected. There are 300 rapes each day which is 26 times higher than in Japan. Thirty-five percent of women who visit emergency wards do so because of violence-related causes. Who pays for the treatment of "crack" babies and AIDS afflicted drug users? We spend more on treating these socioeconomic ills than preventing them. I do not have to tell you about motor vehicle accidents and gun-shot injuries. Even school children settle their differences with guns. Our emergency wards are filled with victims of violent crimes. The United States has the highest per capita incidence of these violent crimes and is considered the homicide capital of the civilized world.

This generates heavy health care costs. We have better laws controlling ownership and operation of a motor vehicle than a firearm, including a hand gun. Another factor contributing to rising health care costs is our lifestyle. We consume more fat that the rest of the world (except Denmark and Belgium) and have the highest incidence of heart disease. Smoking related illnesses add about $65 billion a year and alcohol and drug abuse about $110 billion to our national health care cost. Politicians won't tell the public about these disastrous life-styles costing so much, but we the hospitals and the doctors are doing our best to inform the public. How many health insurance companies add a line item to their premium structures indicating additional costs for providing care for those who smoke and drink and use drugs? None, I dare to say. As we dare to tell the truth about the cost of health care, let us remember what Woodrow Wilson said: "I used to be a lawyer but now I am a reformed character." So we have to talk about tort reform. Product liability and medical malpractice reforms are urgently needed. The lawsuit industry is completely out of control. Liability insurance and indirect costs associated with it add about $100 billion, according to Peter Huber, a senior fellow at the Manhattan Institute and author of the book *Galileo's Revenge: Junk Science in the Courtroom*. Rand Corporation's Institute for Civil Justice estimates that the liability cost in the U.S. is 15 times greater than Japan and 20 times greater than in Europe. Cases filed in state and federal courts have jumped nearly 12 times in the last two decades.

Corporate America has joined the bandwagon in bashing the hospitals and doctors for the rising health care costs even though they know the real reasons for the same. But what they fail to tell the workers is the truth. AMA's Center for Health Policy Research is disputing their claim. They reviewed the tax returns of the major corporations and found out that the employee benefit program including the health care cost made up only 1% of the total deductions. Health benefits were one of the smallest expenses. Corporations spend less than half as much on benefits as they did on compensation for their officers. (Internal Revenue Services, Statistics of Income Bulletin, Fall 1991) The reason the corporations spend so much for health care is to compensate for the other uncompensated care given to society in cost shifting that goes along with it. Even after all that, the corporations and the workers pay the lowest federal and state taxes in the industrialized countries.

Now let us review the paperwork, the hassle factor, and the utilization and review mechanisms that have zapped the little bit of energy the health profession had. It is estimated that $7 billion a year will be used for utilization and review activities. Doubts are growing about the basic premise of this new industry that it will lower medical costs. (Wall Street *Journal,* 28 July 1992) The burdens imposed on doctors and hospitals are increasing the total cost of medical care. It has been pointed out that utilization and review, and other similar programs, have required an army of bureaucrats to eliminate only modest amounts of unnecessary care.

As you can see, I have outlined some of the causes of rising health care cost. But it is definitely not because of doctors. Blaming the doctors and the hospitals for rising health care cost is like blaming the police for increasing crime. That is why we have to proclaim the truth. We have to let the public know that we, the physicians, are the core of the health delivery system. We are their best advocates. We know more about the health care system than everyone put together. We have to start informing the public that global budgeting and health care ceilings and expenditure targets mean rationing. Socialized medicine is good if we are willing to accept rationing that goes with it. We can not provide everything for everybody at all times without paying for it. Let us proclaim the truth about health care; that is but the beginning. Let us get involved in the political process of health care so that it is not taken over by those with a different agenda who want to control us in the name of cost containment.

Sultan Ahamed, M.D.
President

REFLECTIONS ON MEDICINE

Real Reform or Just Tinkering?

ROBERT U. MASSEY, M.D.

In the 1950 *Year Book of Medicine* a note on page 277 referred to a "recent report of Wynder and Graham (*JAMA* 143:329-336, May 27, 1950) [that] contains data suggesting that excessive tobacco smoking may be an important factor" in bronchogenic carcinoma. J. Burns Amberson, the editor of the "Chest" section, noted, "The evidence is not accepted generally without many qualifications indicating the need for better proof." He included tobacco as but one among several irritating substances, including mineral oil and asbestos. In those days this *Journal* carried cigarette ads showing doctors smoking. I knew few physicians in the 1940s and 1950s who did not smoke; I thought them odd, assumed they had some peculiar tobacco sensitivity, or were religious fanatics who also eschewed card playing and the movies.

Consider how in the space of four decades we have changed, abandoning a pleasurable habit that seemed a harmless, almost essential, part of our social lives (and solitary lives as well), transmuting it into a foul-smelling, life-threatening addiction, a sin in the eyes of anti-smoking enthusiasts. Smoking has been on the way out for a decade or more, having been pushed further and further toward the outer edge of acceptable behavior. Attitudes, even those that seem fixed and timeless, do change, not abruptly but often imperceptibly, yielding to continuously applied pressure. Formerly accepted social norms become, first anachronisms, then patently abnormal. *Mores* change with the *tempora*, even for those of us who deny moral relativism. For other examples, all within living memory, consider abortion and homosexuality, both criminal in 1950 when racial prejudice was tacitly, even openly, accepted.

Attitudinal changes that affect a whole society may be driven by some underlying ebb and flow of the collective unconscious; or more likely may reflect less mysterious forces such as population growth, demographic mix, and technological change. Whatever prime mover drives changes in attitudes, manners, and values, its effectiveness depends on language, a facility unique to our bright and brainy species and now communicated with the speed of light.

Health care reform is in the air in this new year and new administration—as indeed it has been five or six times since 1917. This time the pressure is on and something is bound to happen; if history can be relied on at all, odds are that that something will work badly, be counterproductive, flashy but trivial, or produce some nefarious effect that will go unrecognized for a decade.

> Perhaps all structural tinkering is doomed to fall. The root causes of the problem are the increase in demand and the explosion of new forms of technology, These make the rationing of health care inevitable and the chief issue we should be addressing publicly.[1]

Setting limits requires a change in attitudes about medical care. It may depend on changing the very language we use, beginning with *health care*, by which we generally mean *sickness care* or *medical care*. It may require a kind of demedicalizing of our society, paying less attention to the expensive, seductive half-way technologies and more to basic research, public health, and personal responsibility for health care. The term "ration" derives from the Latin for "reason," and implies that society must exercise restraint if it is to be fair in a finite world. It may even suggest that other societal goods, such as education and the environment, be put ahead of medical care which is, after all, but one means to "life, liberty, and the pursuit of happiness." Health care reform is more than tort reform or messing around with the reimbursement business. Giving up our unhealthy addiction to health care will be as hard as giving up cigarettes, but it may be just as necessary.

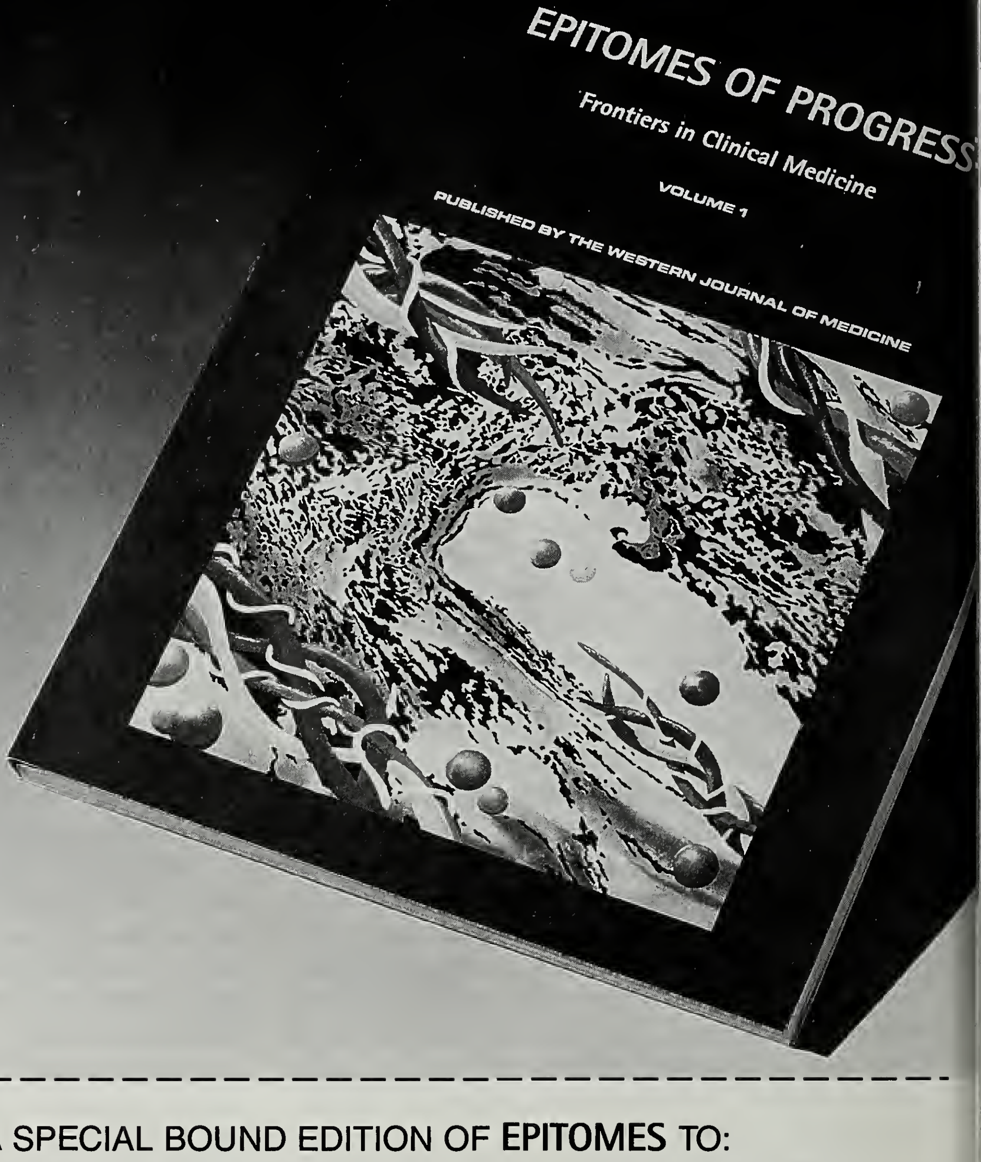
REFERENCE

1. Linton AL: Sounding Board. *N Engl J Med* 1990; 322:198-9.

ROBERT U. MASSEY, M.D., Professor Emeritus, Division of Humanistic Studies in Medicine, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

epitome

i-ˈpit-ə-mē\ *n* [L, fr. Gk *epitomē*, fr. *epitemnein* to cut short, fr. *epi-* + *temnein* to cut — more at TOME] (1520) **1 a** : a summary of a written work **b** : a brief presentation or statement of something **2** : a typical or ideal example: EMBODIMENT (the British monarchy itself is the ~ of tradition — Richard Joseph) **3** : Plural — *Epit-o-mes* — A regular FEATURE of *The Western Journal of Medicine*, now available in a single, bound volume.


EPITOMES OF PROGRESS
Frontiers in Clinical Medicine
VOLUME 1
PUBLISHED BY THE WESTERN JOURNAL OF MEDICINE

Each set of **EPITOMES** is a concise and clear review of the important advances in a medical specialty, chosen and written by experts in that specialty. Now, for the first time, a special bound volume of **EPITOMES** is available. For only $19.95 you can have all the latest advances in 24 different specialties in one attractively designed volume. An excellent reference for your library. A useful gift. Hurry! Supplies are limited. Order a copy today.

YES, SEND A SPECIAL BOUND EDITION OF **EPITOMES** TO:

The Western Journal of Medicine

☐ Yes, send me _____ copy(ies) of the Epitomes book at $19.95 each.

☐ I want to receive Epitomes every month. Please enter my one-year subscription to *WJM* for only $40.00.

For additional information, call (415) 882-5179.
Please mail this coupon with enclosed payment to:
Circulation Department
P.O. Box 7602, San Francisco, California 94120-7602

Name ______________________
Street ______________________
City ______________________
State ____________ Zip ____________
☐ Check enclosed Charge my: ☐ VISA ☐ MasterCard
Card Number ______________________
Expiration Date ______________________
Signature ______________________

Ends and Means in Organ Transplantation: The Anencephalic Donor

JOSEPH M. HEALEY, J.D.

A disproportionately large number of troublesome controversies in health policy occurs at either the beginning or the end of the life of the person, Among the most prominent issues at the beginning of life are; (1) the ethical and legal status of the human being during the process from conception, through gestation and birth, and into childhood; (2) the precise point at which responsibilities and duties are owed to the emerging life; and (3) the criteria used to justify such judgments. Among the most prominent issues at the end of life are: (1) the level of support owed to a dying patient; (2) the point at which obligations cease because the patient is no longer alive; and (3) the criteria used to determine that death has occurred. A few situations involve issues from both the beginning and the end of life. Care of the anencephalic infant is one such example.

A recent case decided by the Supreme Court of Florida, *In re T.A.P.C.,*[1] involved many of these issues from both the beginning and the end of life. Further complicating this case was the additional set of issues involved in organ transplantation. The parents of an anencephalic infant sought to have their daughter declared dead shortly after her birth to assure that her organs would be in the best possible shape for transplantation. Anencephaly is a severe birth defect of the central nervous system characterized by the congenital absence of significant portions of the brain, skull, and scalp. The Court relied upon a recent task force on anencephaly[2] to describe its four essential criteria; "(1) a large portion of the skull is absent; (2) the scalp, which extends to the margin of the bone, is absent over the skull defect; (3) hemorrhagic fibrotic tissue is exposed because of defects in the skull and scalp; and (4) recognizable cerebral hemispheres are absent."[1(p2)] The mother learned of the anencephaly near the beginning of her eighth month of pregnancy. She continued to carry the pregnancy to term and consented to a caesarean section with the clear intent that her infant's organs would be less damaged and available for transplantation. The parents expressed their hope that some good would come out of this tragedy in the form of a gift of life to others who would benefit because of the transplanted organs. The infant's health professionals refused, expressing the fear that such actions were prohibited by the Florida "brain death" statute and that civil or criminal liability might result if the wishes of the parents were carried out. The parents brought suit to clarify the legal boundaries of their rights and responsibilities. The trial court refused to grant permission for the declaration of death, citing the Florida statute as requiring the satisfaction of criteria that would generally not be present in an anencephalic at birth. While the appellate court affirmed the trial court's decision, it sought to expedite further review by the Florida Supreme Court.

In its decision, a unanimous Court reviewed existing Florida law concerning the determination of death. The Court concluded that the common law of Florida recognized a cardiopulmonary definition of death, emphasizing the cessation of breathing and of heartbeat, without the possibility of resuscitation. To supplement that definition, statutory law acknowledged "whole-brain death" as an acceptable legal standard for determining that death had occurred in cases when cardiopulmonary function is being maintained through the use of life support systems such as a respirator. Since this alternative did not apply to the case at hand, the Court concluded that the appropriate standard must be the common law, cardiopulmonary standard. Since the evidence demonstrated that the infant's heart was beating and that she was breathing, she was not dead and her organs could not be removed at this time. The justices acknowledged the parents' plight:

JOSEPH M. HEALEY, J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

Address for correspondents: Joseph M. Healey, J.D., Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington, CT 06030-1910.

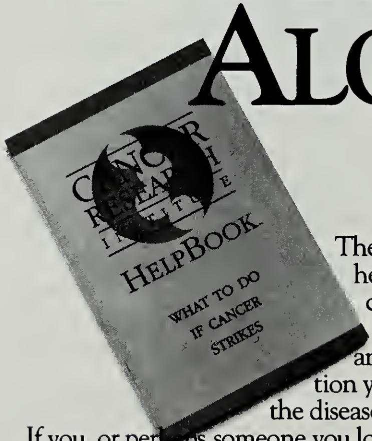
> We have been deeply touched by the altruism and unquestioned motives of the parents of T.A.C.P. The problem we as a Court must face, however, is that the medical literature shows unresolved controversy over the extent to which anencephalic organs can or should be used in transplants.[1(p6)]

The Court refused to grant the parents' wishes about the transplantation of their infant's organs. This case raises policy questions about the important end of increasing the availability of organs for transplantation, while asking whether there are means to that end that exceed reasonable bounds. Discussion of the case will continue in next month's column.

REFERENCES

1. No. 79-582 (Fla. Sup. Ct., Nov. 12, 1992).
2. The Medical Task Force on Anencephaly. The infant with anencephaly. *N Engl J Med* 1990;322:669-73.

IF THE THOUGHT OF CANCER SCARES YOU, YOU'RE NOT ALONE.



The Cancer Research Institute is here, with help — and hope. Just write us for your free* copy of our *HelpBook*.

We've written it so millions of men and women like you can have the information you need to make decisions and deal with the disease.

If you, or perhaps someone you love, has been diagnosed with cancer, the *Cancer Research Institute HelpBook* can tell you about your choices, how to seek excellent care, and how to continue with a complete and productive life.

Even if cancer is not part of your life, you'll find it a valuable reference book to have on hand should things change.

Write today. *Cancer Research Institute HelpBook*, F.D.R. Station, Box 5199, New York, NY 10150-5199.

CANCER RESEARCH INSTITUTE

* *$2 per copy for postage and handling.*

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgment), provided that they are submitted in duplicate, signed by all authors, typewritten in double spacing, and do not exceed 1½ pages of text (excluding references). They should not duplicate similar material being submitted or published elsewhere. Letters referring to a recent *Journal* article should be received within six weeks of the article's publication.

DEFENSIVE MEDICINE

To the Editor: Dr. Ahamed should be commended for his comments on tort reform in the September issue. The present system compels physicians to practice defensive medicine which is primarily responsible for the high cost of medical care.

Most physicians routinely practice defensive medicine in hopes of discouraging any allegations of neglect in the event of an unfavorable outcome because if litigation does occur, regardless of whether the verdict is guilty or not guilty, the physician knows that being a defendant in a malpractice suit is a very painful ordeal.

It is unbelievable that the medical establishment has not challenged the legal profession more openly for its exploitation of the legal vulnerabilities which all physicians assume when they treat patients.

Edward J. Volpintesta, M.D.
Bethel

COOPERATION AND COMPROMISE

To the Editor: The health care cost problem could be solved instantly if three groups give up perks. Doctors relinquish fee-for-service. Lawyers null the medical malpractice industry. Cover all citizens for standard care and eliminate administrative costs.

If the vested interests involved all give up a little bit, if there is cooperation and compromise, if there is patriotism enough to unite and help others instead of self-serving only, we could easily do it. This is an opportunity for all involved to show humanity.

Capitalism looks obscene if a respectable, responsible segment of the population has no health insurance coverage. It is only a small matter of rearranging our priorities! Universal care coverage need only eliminate the profit motive replacing it with the higher motive.

We cannot be crass enough to reject this limited amount of socialism in a single area. Removing marketplace motivation from only a tiny portion of our society would show nobility of character. But such nobility is a minimum necessity to preserve our nation and our culture and our quality of life!

Sidney Vernon, M.D.
Willimantic

YOCON®
YOHIMBINE HCI

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

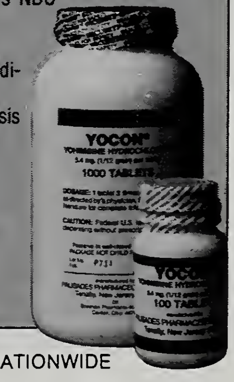
Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



AVAILABLE AT PHARMACIES NATIONWIDE

PALISADES PHARMACEUTICALS, INC.
219 County Road
Tenafly, New Jersey 07670
(201) 569-8502
1-800-237-9083

This Month's Reading in Review

TIMOTHY B. NORBECK

- Supporters of a single-payer health insurance system point to the low administrative costs (2.1%) of Medicare compared to private insurance companies. . . . This difference is due mainly because Medicare's Part A is compulsory, eligibility for it is determined primarily by age, and there is only one standard benefit package. . . . The program has no sales, marketing, risk-assessment, or insurance-commission costs. . . . Also, costs are lower because a much higher proportion of its submitted claims are transmitted electronically.
- The 7.4% growth in Medicare spending, from $109.7 billion in fiscal year 1990 to $117.8 billion in 1991, reflects a rising number of claims submitted. . . . Medicare beneficiaries increased by about 700,000 (or 1.9%) over that year while the number of claims rose nearly 11% to reach over 600 million. . . . (NOTE: Such an increase in claims was to be expected since physicians were required in 1991 to file patient claims)
- The General Accounting Office (GAO) reported in 1989 that it had found more than $150 billion in waste, fraud and financial mismanagement in the federal government.
- Over the past 100 years, Ohio and Missouri have truly been a bellwether and microcosm of the national mood in presidential elections. . . . In the 24 elections this century, the two states have voted for the winner 22 times—a success rate matched by no other state.
- The amount of wages subject to the Social Security and Medicare payroll taxes in 1993 will be $57,600 and $135,000 respectively, compared to $55,500 and $130,200 for 1992. . . . The tax rate paid by employers and wage earners will remain unchanged next year at 7.65% of wages—6.2% for Social Security and 1.45% for Medicare. . . . For self-employed individuals, the rate will remain at 15.3% of earnings—12.4% for Social Security and 2.9% for Medicare.
- According to a November Harris poll for MetLife, 63% of policy makers "believe high-tech medical care must eventually be rationed". . . . A Gallup survey indicates that only 20% of the public believes there should be limits on care.
- A couple retiring in 1991, with one wage earner and who had paid average Medicare taxes since 1966, would have contributed a total of $8,480. . . . The present value of future Part A benefits for such a couple is estimated to be $86,320, or more than 10 times the amount they paid into the trust fund. . . . For Part B, retirees receive benefits that average four times their premiums.
- German researchers, reporting last month to an American Heart Association meeting in New Orleans, said that the risk of employees having a heart attack on Monday is 40% higher than on the other days of the week. . . . Scientists speculate that heading back to the office may severely increase mental and physical stress and that heart problems might also be triggered by heavy weekend drinking. . . . There is no explanation, however, on why Thursday is the second riskiest day for worker heart attacks.
- "Only all-out reform has a chance. Health care special interest groups have enough power to block tinkering. We've got to get them falling back on their heels with a broad program that changes everything." Rep. Jim Cooper (TN), and a leader in the Conservative Democratic Forum, on health reform.
- According to a study at Georgia State University, people eat more in autumn (11%-14% more) than in any other season. . . . A possible explanation is that humans, like other animals, are conditioned to overeat in autumn to prepare for a lean winter.
- Medicare's end-stage renal disease benefit is unique; it is the only instance where a diagnosis provides the basis for Medicare benefits for persons of all ages.
- Only in America: When a 66-year-old California woman was diagnosed with cardiopulmonary obstructive disease, her physician prescribed an electric machine that distributes medication through a breathing device and which costs a mere $119. . . . The Medicare carrier, however, refused to pay for the purchase of the machine and advised the patient to rent it for $43.86 a month. . . . Inasmuch as the patient has had the breathing device for 37 months, Medicare has now cumulatively spent more than $1,200 on an item that could have been purchased for $119.

TIMOTHY B. NORBECK, Executive Director, Connecticut State Medical Society.

BOOK REVIEW

Handbook of Clinical Dietetics

Second edition. The American Dietetic Association. 575 pp., illustrated. New Haven, Yale University Press, 1992. $60. ISBN 0-300-05218-9.

At a glance, the title of this book may bring to mind the notion that this is just another diet manual. The reader will be pleasantly surprised at the practicality and thoroughness of this valuable reference book. This is much more than a completion of lists of "Food Allowed" and "Foods Excluded" for a variety of disease states. Rather, for every therapeutic diet reviewed, there are sections covering definitions, purpose of the diet, effects of the diet, related physiology, foods and nutrients, indications for use, possible adverse reactions, contraindications of the diet, clinical assessment, potential drug interactions, implementation and education, priorities for quality assurance, and sample menus.

For a reference book, this is actually enjoyable reading. Each chapter includes an intelligent review of the scientific basis for diet modification in a specific disease state. These sections are well-researched and well-referenced. Areas where current research is either inconclusive or not available are clearly identified.

One unique feature of this resource is the practical manner in which potential drug-nutrient interactions are addressed. For each disease state, the authors provide the nutritional implications of medications commonly prescribed to treat the disorder. This eliminates the need to seek another reference for this information.

The sections dealing with quality assurance priorities are helpful, although they do not discuss specific patient outcome indicators which are required by the JCAHO. These sections do serve to highlight areas where clinical dietetic programs may identify possible problems or areas for improvement. This is definitely a step in the right direction.

This handbook also includes three position papers approved by the American Dietetic Association, covering Nutrition Intervention in the Treatment of HIV Infection, Very-Low-Calorie Weight-Loss Diets, and Nutrition Intervention in the Treatment of Anorexia Nervosa and Bulimia Nervosa. There are also chapters covering enteral and parenteral nutrition. These are valuable additions to this handbook, as these are frequently encountered nutritional issues without a specific diet order.

This book certainly meets its intention to "stimulate interest, discussion and research to clarify gaps in clinical diet therapy." I would recommend this book to any health-care professional who is interested in a practical and thorough clinical nutrition reference book.

Donna Chapman, M.S., R.D.
Director of Clinical Dietetics
John Dempsey Hospital, Farmington

Quinnipiac College to Sponsor Conference on 3 March 1993 on "AIDS: Pharmaceuticl Treatment and Vaccine Development"

Quinnipiac College's National Institute for Community Health Education (NICHE) will sponsor its annual conference on Wednesday, 3 March 1993 on the Hamden Campus.

Five leading experts from the medical, pharmaceutical, and public health arenas will present the latest information on AIDS at a panel discussion, which will take place in the Gymnasium, from 9:30 AM to 12 NOON . The panelists are: Gerald Friedland, M.D., Director, Yale-New Haven Hospital Program on AIDS, Moderator; Marcel Rozencweig, M.D., vice president of Clinical Research-Center and Infectious Diseases, at Bristol-Myers Squibb, who will focus his remarks on the pharmaceutical control of AIDS; Franklin Volovitz, chairman and president of MicroGeneSys Inc., who will discuss the efforts to develop an AIDS vaccine; Susan Silliman Addiss, M.P.H., M.Ur.S., Commissioner, Connecticut State Department of Health Services, will address the public health concerns surrounding both the pharmaceutical treatment of AIDS and the potential for vaccine development; and Alvin Novick, M.D., professor of biology at Yale University, will discuss consumer concerns.

The luncheon and keynote address will follow at 12:15 PM in Alumni Hall, at a charge of $15.00 to cover the cost of the luncheon. For further information, call (203) 281-8655.

CSMS PHYSICIAN PLACEMENT SERVICE

The Society maintains the Physician Placement Service as a *free* service to the medical profession, hospitals, and communities in Connecticut.

Opportunities* should be typed, double-spaced copy on letterhead and submitted to CSMS, Physician Placement Service, 160 St. Ronan St., New Haven, CT 06511 (203) 865-0587. These will be published as space permits and will be distributed to physicians making inquiries of such *opportunities.

***Physicians wishing to locate* in Connecticut may call the office requesting opportunities in their specialty. Also, candidates are invited to submit a resume to be kept on file with the Society. An announcement of a physician's availability will be published in two issues of *Connecticut Medicine* as space permits.**

Listing of physicians in the Placement Service does not in any way represent certification by the Society. Investigation of credentials and experience is the responsibility of those seeking applicants for positions.

Announcements on the Physician Placement Service page under Classified Advertising are charged at the regular Classified Advertising rate.

OPPORTUNITIES FOR PRACTICE

AMBULATORY CARE

Single site ambulatory care center in southeastern Connecticut seeking Family/General Practitioner or Internist for full or part-time position. Well rounded medical background desired, including experience in pediatrics and office gynecology. Excellent patient relation skills are required. Send CV or call for further information: W. Larry Beason, M.D., FAAFP, Charter Oak Medical Center, 324 Flanders Road, East Lyme, CT 06333. Telephone: (203) 739-6953.

Full time or part time positions available in ambulatory care offices in Central Connecticut. No on-call duty. Pleasant conditions and staff. Moderate volume practices. Please apply to Medical Director, Newington Medical Care Center, 955 Main Street, Newington, CT 06111.

EMERGENCY MEDICINE

Busy walk-in center seeking full-time or part-time physician, requires general medicine, minor-surgery and urgent-care treatment experience. Wonderful staff, great community, good salary and benefits. Contact: Dr. T. Kandell / Dr. R. Falit, Med-Help Medical Center, P.O. Box 1120, Bristol, CT 06010. Telephone: (203) 584-8900.

EMERGENCY PHYSICIANS, Connecticut—Excellent opportunity opening up in May, 1993 for two emergency physicians. Positions based in emergency department of free-standing satellite, affiliated with a 380 bed community hospital located in beautiful shoreline area. Satellite facility is a unique combination of emergency and acute care medicine which accepts ambulances, has full emergency department privileges, and paramedic base station. Current volume is approximately 29,000 patients per 16 hour day, but we're expanding to 24 hour per day operations in May. Physicians will be based at our satellite, but may also do 10–20% of the shifts in hospital-based ED in Middletown (census 34,000). We seek an EM/FP/IM BE/BC individual, ACLS, ATLS and ED experience required. We offer outstanding compensation package. Interested candidates should send or fax resume to: Michael Saxe, M.D., Medical Director, Middlesex Medical Center, Shoreline, 260 Westbrook Road, Essex, CT 06426; FAX (203) 767-1563, or call (203) 767-0107. An Equal Opportunity Employer.

Emergency Physician—Full time position for board prepared, experienced emergency unit physician at an 85-bed acute hospital in central Connecticut. Current Connecticut license required. Includes involvement in Occupational Health program. Competitive salary and excellent benefit package. Call John Scarfo, M.D. at (203) 276-5210 or send CV to John Scarfo, M.D. Chairman Emergency Medicine, Bradley Memorial Hospital, 81 Meriden Avenue, Southington, CT 06489. EEO/M/F/D/V.

FAMILY PRACTICE

Southwestern Connecticut-Family Practice-Ideal opportunity for motivated and energetic BC/BE Family Practitioner to join solo provider in rapidly growing private Family Practice. If you have been thinking about entering into a solo or private Family Practice this is a unique situation. The groundwork has been laid and with the right individual(s) this practice will develop into a quality Family Practice Group. Open and flexible to discussions regarding the position and expansion of the practice. Please contact via telephone and send C.V. to: Alan T. Falkoff, M.D., 838 High Ridge Road, Stamford, CT 06905. (203) 322-7070.

Two Family Practice group looking for 3rd associate. Traditional family practice (no OB) with good mixture of patients. We admit patients to local hospital that trains family practice residents. Please contact Susanna Thomas, M.D. Telephone: (203) 388-1495.

INTERNAL MEDICINE

INTERNISTS, SUB-SPECIALTY ENDOCRINOLOGY OR GERONTOLOGY DESIRABLE—Alternative practice Models available—solo, group, salaried. Excellent financial arrangements and relocation assistance. Eighty-five bed hospital with multi-specialty staff and extensive diagnostic resources. Several primary care physicians retiring in the near future. Central Connecticut area, 2 hours to New York City or Boston. Send letter and CV to: President, Bradley Memorial Hospital and Health Center, 81 Meriden Avenue, Southington, CT 06489.

MEDICAL DIRECTOR

Medical Director-BE/BC EM, IM, OM, GS for rapidly expanding industrial medicine practice. New state of the art facility with xray, lab, rehab in New Haven area. Competitive package including incentive compensation and malpractice insurance. Send resume to Sam Paul, President of Industrial Health Care, 1080 Dayhill Road, Windsor, CT 06095.

OBSTETRICS & GYNECOLOGY

Full-time or part-time position available for a board eligible or certified OB/GYN at a community health center in Hartford. Knowledge in Spanish is desired. Please send CV to: Dr. Reddy, 81 Overlook Terrace, Hartford, CT 06106, or call (203) 236-0857.

Trends in Breast Cancer Treatment in Connecticut

ANTHONY P. POLEDNAK, PH.D. AND JOHN T. FLANNERY, B.S.

ABSTRACT—Data from the population-based Connecticut Tumor Registry showed an increase in partial mastectomy (PM) from 18.9% for local-stage breast cancers diagnosed in 1983 to 47.3% for those diagnosed in 1990, with no change in axillary dissection. For local-stage cancers 4 cm or smaller in size that received PM, axillary dissection increased from 39.2% in 1983 to 60.8% in 1990, with larger increases for cases < 50 and 50–59 years old, while radiotherapy increased mainly for cases 70–79 and > 80 years old. Among regional-stage cases with positive nodes (but no extension to skin or beyond the breast tissue) and 4 cm or smaller in size, PM increased from 9.4% to 29.1%, but remained lower than that among local-stage cancers. Continued monitoring is needed to assess the full impact of the 1990 NIH Consensus Conference Panel's recommendations for the treatment of early-stage breast cancer.

Introduction

WITH the publication (in 1985) of clinical trials showing similar prognosis with partial mastectomy (PM) or "breast conserving surgery" *vs* more extensive surgery,[1,2] PM has become the recommended procedure for "early" breast cancer. PM was also regarded as "preferable" by the National Institues of Health (NIH) Consensus Panel of 1990 which also recommended axillary lymphnode dissection and postoperative radiotherapy for "early-stage" breast cancers, or stages I (2 cm or less in size, negative axillary nodes) and II (2–5 cm, with or without positive nodes) in the American Joint Committee on Cancer (AJCC) system.[3] Data from the National Cancer Institute's (NCI) Surveillance, Epidemiology and End Results (SEER) Program showed an increase in PM and a decline in mastectomy for stage I and II cancers diagnosed between 1983 and 1987, although PM was still not predominant.[4] It has been suggested that the use of axillary dissection during surgery for breast cancer may be declining in some countries because of increasing use of PM as well as changes in the size of the invasive breast cancers detected.[5,6] This paper examines time trends in the use of these treatments for breast cancer in Connecticut residents.

Materials and Methods

Data were analyzed on type of surgery, use of axillary lymphnode dissection during surgery, and postoperative radiotherapy in the first course of treatment among all female primary breast cancers diagnosed in 1983–90 and reported to the population-based Connecticut Tumor Registry (CTR), which is part of the SEER Program. Data were considered complete for cases diagnosed through 1989. Although reporting of cases diagnosed in 1990 may be incomplete, the total number of cases already exceeds that for 1989 and the importance of monitoring time trends in treatment adds impetus to using preliminary data for 1990 cases.

A coding scheme for clinical stage at diagnosis has been used consistently in the CTR since its inception in 1935. The stages are in situ, local, regional (ie, involvement beyond the breast into surrounding tissues, and/or regional lymph node involvement), and metastatic (ie, direct extension to "remote" organs and/or distant metastases); this scheme is similar to that used in reports from the SEER program[7,8] and the American College of Surgeons' national surveys of breast cancer.[9] Of the 20,412 breast cancers diagnosed among Connecticut residents in 1983–90, 10,415 or 51% were diagnosed at the local stage, with an increase from 47% (993/2,113) in 1983 to 52% (1,490/2,863) in 1990. Some 3,885 cases were diagnosed at the regional stage without involvement of skin and without direct extension to tissues beyond the breast (ie, node-positive cases only), with a decrease from 24.3% for 1983 to 15.5% for 1990. Cases ascertained by autopsy or death certificate only were excluded from analyses of treatment. Tumor size, which was of interest because clinical trials involved cancers 4 cm or smaller in size,[1] was coded for 85.4% of local-stage cancers and 90.3% of regional-stage cancers diagnosed in 1983–90.

ANTHONY P. POLEDNAK, PH.D. AND JOHN T. FLANNERY, B.S., Connecticut Tumor Registry, Connecticut Department of Health Services, Hartford.

Data in this report were collected under the auspices of Contract N01-CN-05226 between the National Cancer Institute and the Connecticut Department of Health Services.

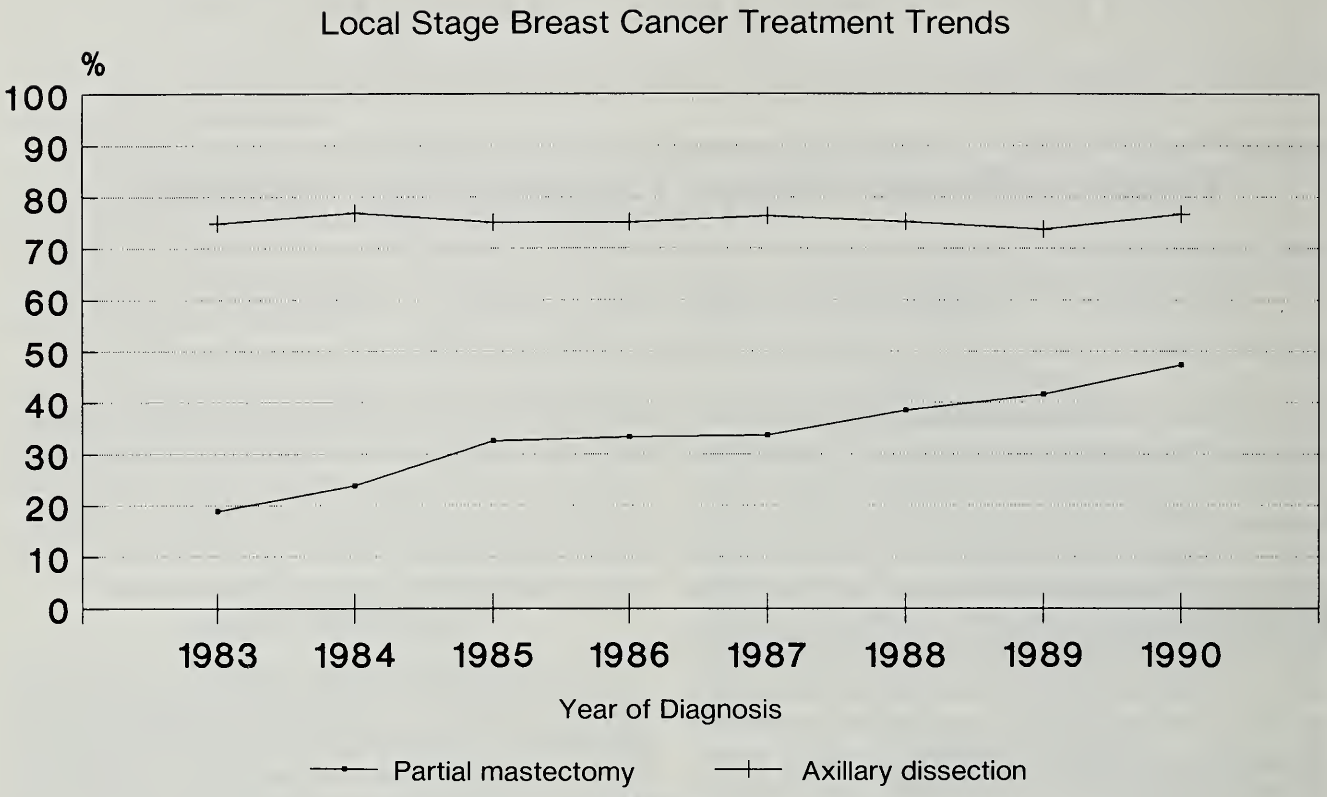


Figure 1

Proportions of local-stage breast cancers receiving partial mastectomy and axillary dissection, by year of diagnosis, in Connecticut.

In the SEER Program the first course of treatment is defined as any treatment received within four months after the initiation of therapy. Accuracy of abstracting/coding SEER data on radiotherapy and site-specific surgery is excellent, according to the results of NCI monitoring;[10] SEER data on chemotherapy are incomplete and are not included in this report. The SEER definition of lymph node dissection, as involving four or more nodes, is supported by the Danish Breast Cancer Cooperative Group's report that risk of early ipsilateral axillary relapse was only 2% in patients with at least three negative nodes at initial surgery,[11] although other data suggest that five or more, or 10 or more, nodes removed (and negative) may be more predictive.[12]

Type of breast-cancer surgery was coded according to the SEER system, which identifies partial mastectomy (PM), including segmental mastectomy, lumpectomy, quadrantectomy, tylectomy, wedge resection, nipple resection, excisional biopsy, or PM not otherwise specified. Within the PM group there are separate codes for cases with and without axillary lymphnode dissection. Some 3,616 of 10,415 local-stage cancers (34.7%) diagnosed in 1983–90 had PM, and 50.5% of the 3,616 PM cases included axillary dissection. Other types of surgery (ie, non-PM) for local-stage cancers included: subcutaneous mastectomy (with or without axillary dissection) (n = 20 or 0.2% of 10,415); total (simple) mastectomy (breast only, without lymph node dissection) (n = 472 or 4.5%); modified radical mastectomy (n = 6,007 or 57.7%); and radical mastectomy (30 or 0.3%). Because modified radical mastectomy was much more common than the other (non-PM) types of surgery, most non-PM surgery involves axillary dissection. The remainder of the 10,415 local-stage cancers involved unspecified type of surgery (n = 24 or 0.2%) and no surgery (n = 246 or 2.4%).

Regional stage cancers (as defined above) showed a different pattern of treatment, with only 14.7% (570/3,885) receiving PM and 82.4% receiving modified radical mastectomy.

Results

The proportion of local-stage cancers treated with PM increased for cases diagnosed in 1984 and (especially) 1985 *vs* 1983, leveled off for 1986 and 1987, and increased thereafter. The proportion receiving PM increased from 18.9% (188/993) for 1983 to a level 2.5 times higher (ie, 705/1,490 or 47.3%) for 1990, while there was no change in the proportion receiving axillary dissection (ie, 74.9% for 1983 and 76.7% for 1990) (Fig. 1). Further analyses concentrated on local-stage cases 4 cm or less in size, or the size group involved in clinical

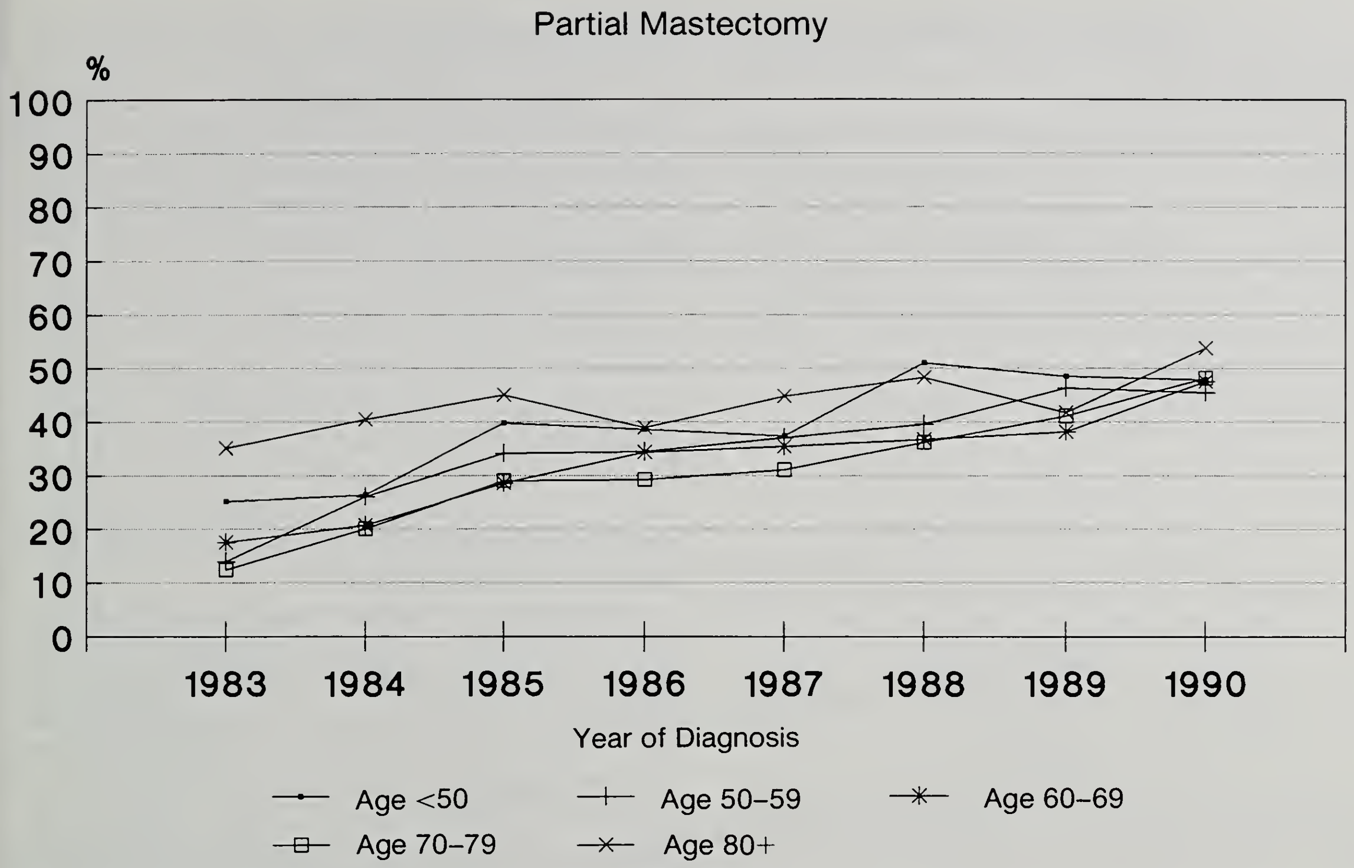


Figure 2

Proportion of local-stage breast cancers (4 cm or less in size) receiving partial mastectomy, by age at diagnosis and year of diagnosis.

trials.[1] Among cases diagnosed in 1983 PM was most frequent for cases > 80 and < 50 years old, and increases over time were evident for all age groups (Fig. 2). Proportional increases over time were smaller for ages < 50 and > 80 years, so that age differences in PM (Fig. 2) were smaller for cases diagnosed in 1990 (χ^2 test, 4 degrees of freedom, $P = .559$) *vs* 1983 ($P < .0001$). Within each age group large increases in PM were evident for cases diagnosed in 1985, or the year in which the results of clinical trials were published.[1] There appeared to be a leveling off for cases diagnosed in 1986–87 and additional increases for 1988–1990 cases for all ages combined (Fig. 1) and for most age groups (Fig. 2).

The increase in use of PM over time was not due to the increase in smaller size cancers. While PM varied by tumor size the increase was similar for local-stage cancers 2 to 4 cm (ie, from 15.8% for 1983 to 35.8% for 1990, or 2.3 times the proportion for 1983) and for those < 2 cm in size (ie, from 23.9% for 1983 to 54.3% for 1990, or also 2.3 times the 1983 level). The latter figures for < 2 cm were similar to those for < 1 cm (ie, 25.8% for 1983 and 56.3% for 1990).

The lack of decline in axillary dissection, despite the large increase in PM (Fig. 1), was explained by an increase in axillary dissection within the PM group. For cancers 4 cm or less in size, axillary dissection within the PM group increased from 39.2% (58/148) for 1983 to 60.8% (377/620) for 1990. Increases were greatest for ages < 50 and 50–59 years, resulting in greater age differences for cases diagnosed in 1990 *vs* 1983 (Fig. 3A).

Within the PM group the proportions receiving postoperative radiotherapy were already high in the younger age groups in 1983 and showed no increases (Fig. 3B). Sample sizes were small for the earliest years of diagnosis, especially for younger age groups, reflecting the lesser use of PM in earlier years of diagnosis. For ages 70–79 and 80 or more years the proportion receiving radiotherapy increased between 1985 and 1986, with little or no further increase (except perhaps for cases 80 or more years old diagnosed in 1990). By 1990 age differences had become somewhat smaller, although radiotherapy was still uncommon for age 80 or more years and the association with age was statistically significant ($P < .0001$).

Within local-stage cancers 4 cm or less in size diagnosed in 1983–90 there was no evidence for lesser use of PM for blacks (133/336 or 39.6%) *vs* whites (2,880/7,931 or 36.3%), or for lesser use of post-PM radiotherapy

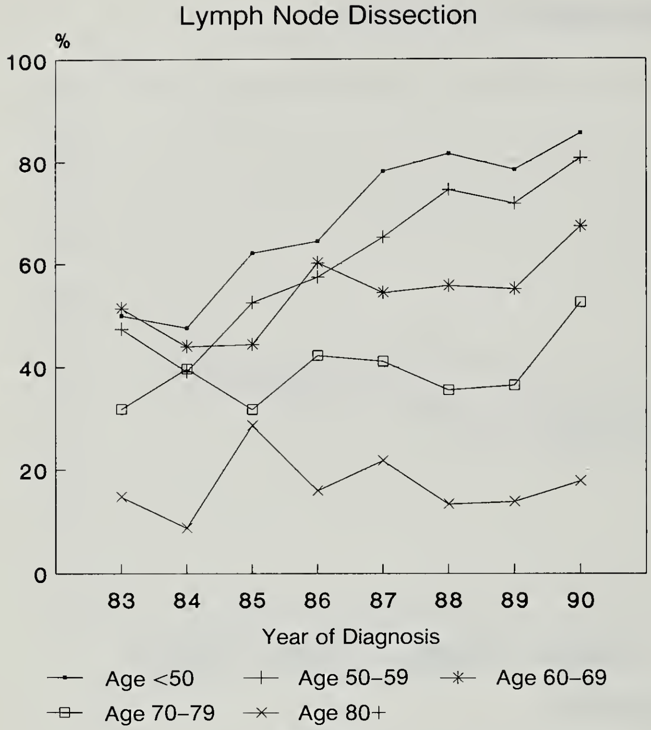


Figure 3A

Local-stage breast cancers 4 cm or less in size receiving partial mastectomy: Proportion receiving axillary dissection by year of diagnosis.

among blacks (88/133 or 66.2%) *vs* whites 1,674/2,880 or 58.1%). Increases in use of PM over time were apparent for both races, and for cases diagnosed in 1990 differences were small (ie, 590/1,226 or 48.1% for whites and 27/50 or 54% for blacks).

Among regional stage (ie, node-positive without extension) cases with tumors 4 cm or less in size PM increased from 1983 to 1985 but then declined slightly in 1986 and 1987 and increased thereafter, especially in 1990 (Fig. 4). The proportion for cases diagnosed in 1990 was three times as high as that for cases diagnosed in 1983 (ie, 39/414 or 9.4% for 1983 and 102/351 or 29.1% for 1990). PM was significantly more frequent for local *vs* regional stage cancers in each year (Fig. 4). Almost all regional stage cancers (positive nodes) were coded as having had axillary dissection (ie, 96.5%), which led to the diagnosis of node-positive cancer, with little change over time. Some 57% of PM cases were coded as receiving radiotherapy, but the proportion varied for each year of diagnosis with no clear trend (data not shown). There was no evidence for lesser use of PM or post-PM radiotherapy among blacks *vs* whites, ie, 17.8% (27/152) *vs* 15.9% (450/2,836) for PM, and 63% (17/27) *vs* 56.7% (255/450) for post-PM radiotherapy.

Discussion

Limitations of Data.—Although the CTR is a population-based resource, underreporting of certain treatment modalities may occur. Hospital registrars may have difficulty obtaining information on postoperative radiotherapy.[13] NCI's *Patterns of Care Study*, involving intensive collection of treatment data for samples of cases with selected cancers from each SEER registry, suggests some under-ascertainment of radiotherapy for breast cancer in the CTR;[14] it is not known if underascertainment varies by calendar year or age.

The American College of Surgeon's National Cancer Data Base (NCDB) is not population-based and in 1991 included only two hospitals in Connecticut; incompleteness of data on treatment is also possible.[15] Therefore, continued reliance may have to be placed on the CTR for assessing time trends in cancer treatment (at least for surgery and radiotherapy) in Connecticut, especially since large number of cases are required for analyses by age, race, or other subgroups.

Type of Surgery, Axillary Dissection and Radiotherapy.—Time trends for PM within local-stage cancer in Connecticut were consistent with those reported for all SEER areas for stage I or stage IIA cases diagnosed from 1983 to 1987.[4] In Connecticut age differences in PM have decreased over time, although for cases diagnosed in 1990 the proportion remains slightly higher for patients age 80 or more years. Only for cases 80 or more years old has PM reached 50% (ie, 53.7%) in 1990, so that the majority of local-stage cases still did not receive PM. Among PM cases, however, use of both axillary dis-

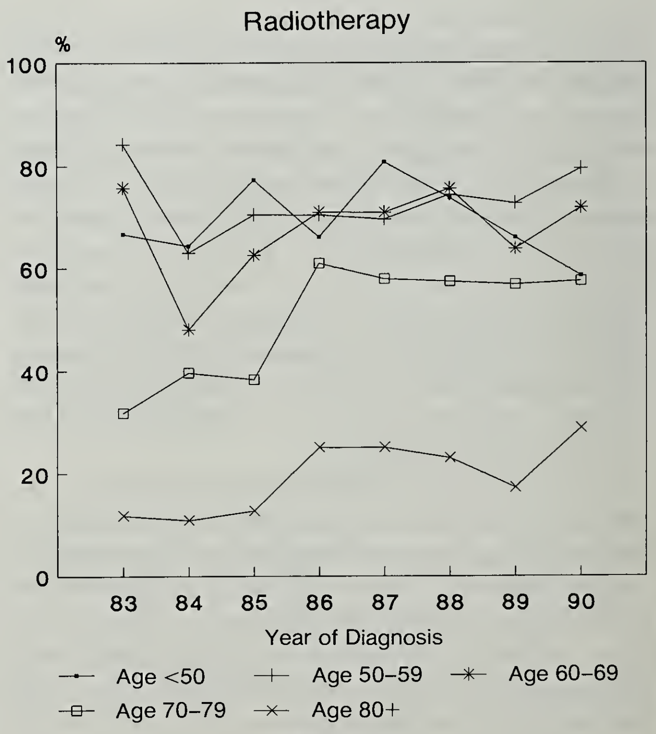


Figure 3B

Local-stage breast cancers 4 cm or less in size receiving partial mastectomy: Proportion receiving post-PM radiotherapy by year of diagnosis.

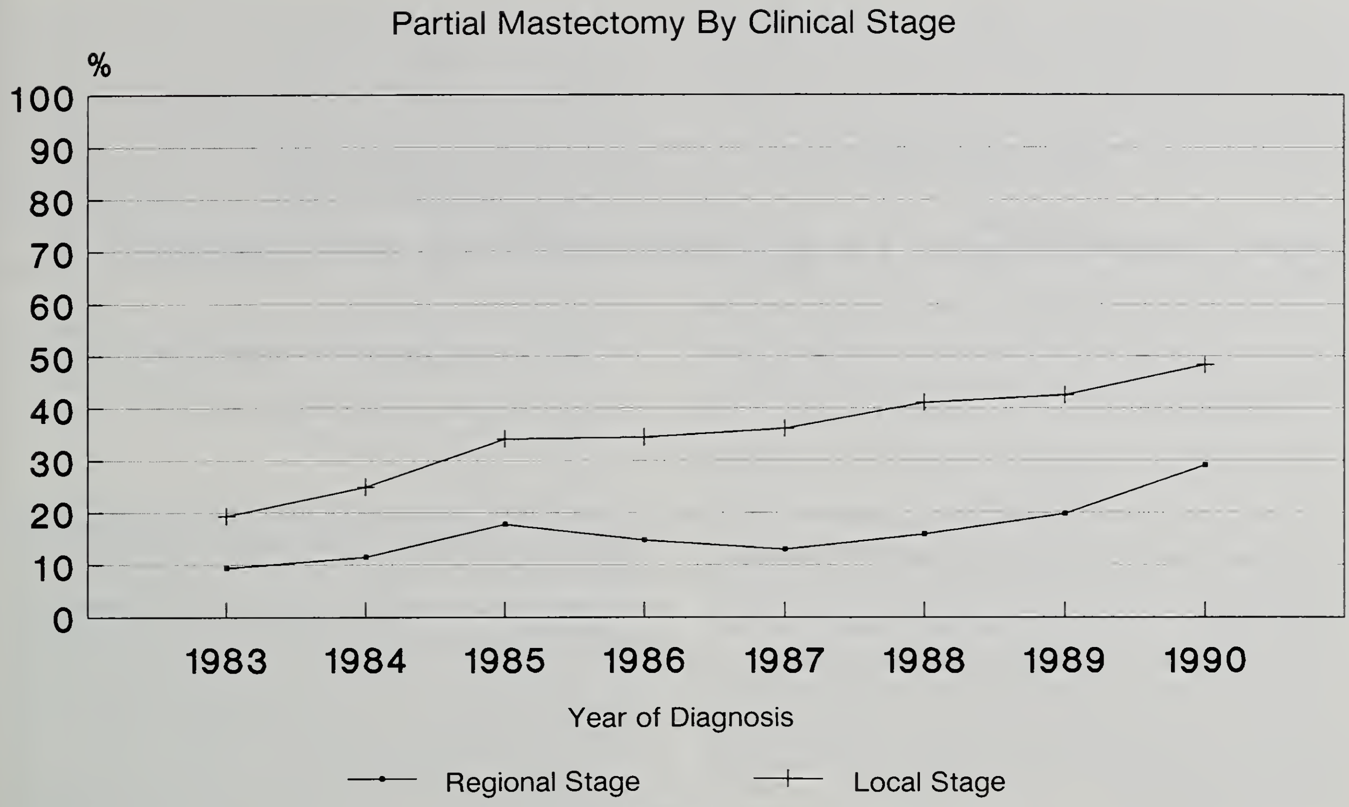


Figure 4

Proportion of cases (with tumors 4 cm or less in size) receiving partial mastectomy by clinical stage and year of diagnosis: "regional" stage includes node-positive cases without extension to skin or beyond the breast (see text).

section and radiotherapy reached about 60–80% for ages < 70 years in 1990.

Despite the large increases in the use of PM, among all local-stage cases there was no evidence for a decline in use of axillary dissection (Fig. 1). This was due to an increase in the proportion of PM cases receiving axillary dissection. The lesser use of axillary dissection at older ages (especially at age 80 or more years),[16] was confirmed in this study. Age differences in the use of axillary dissection were apparent for each year of diagnosis and the association with age has become stronger since 1983 (Fig. 3A). The decrease in post-PM radiotherapy with increasing age was consistent with that in other reports, including a study of cases diagnosed in 1983–89 in another SEER area (ie, Seattle - Puget Sound, Wash.),[17] but in Connecticut age differences had become somewhat smaller for cases diagnosed in 1990 (Fig. 3B). The role of comorbidity factors, and both physician and patient input in treatment decisions, as explanations for age differences cannot be assessed with the SEER data base. Marked increases over short time periods in the use of PM (Figs. 1 and 2), and in the use of axillary dissection among younger PM cases (Fig. 3A), and post-PM radiotherapy among older cases (Fig. 3B), cannot be explained by changes in comorbidity. These changes immediately following the publication in 1985 of the results of clinical trials clearly suggest an effect on practice patterns, as noted by others.[17] Previous reports have not included cases diagnosed in 1990, for which an effect of the 1990 NIH Consensus Conference panel recommendations on treatment of early-stage breast cancer might begin to be detected. The present findings show relatively large increases in PM (Figs. 1 and 4) and axillary dissection within PM cases (Fig. 3A) for cases diagnosed in 1990; the evidence for post-PM radiotherapy is less supportive, except perhaps for cases 80 or more years old (Fig. 3B). In Connecticut there was no evidence for black-white differences in treatment of local-stage breast cancers by PM, axillary dissection, or post-PM radiotherapy. In contrast, Detroit black women with local-stage breast cancer treated at small hospitals in 1985–87 were less likely than white patients to receive PM and post-PM radiotherapy.[18] Differences in use of adjuvant chemotherapy and hormones cannot be adequately assessed with SEER data. The NCDB, however, found no differences in type of systemic therapy for black or low-income (*vs* all) breast cancer patients diagnosed in 1988, although possible underascertainment of systemic therapy was noted.[15]

In Connecticut PM is used less often for regional stage (node-positive without extension only) *vs* local stage cancers 4 cm or less in size, and while PM has increased over time for both stages the difference by stage has persisted (Fig. 4). These findings are consistent with those reported in the Seattle area for stage I *vs* stage II (AJCC criteria), and could reflect an overly cautious interpretation of the results of clinical trials by clinicians.[17]

Conclusion

Monitoring of treatment patterns should continue in Connecticut in order to assess the effect of the 1990 NIH panel's recommendations. Information relevant to treatment decisions continues to be published, including findings from follow-up studies indicating less difficulty with clothing and body image (albeit no difference in quality of life)[19] for PM *vs* mastectomy patients, and no difference in fear of recurrence.[20] Studies are needed on those factors that influence treatment decisions by physicians and patients[17,21] in order to interpret time trends in PM *vs* mastectomy, and to explain age differences in axillary dissection and post-PM radiotherapy. The effect of treatment cost (if any) should be explored; although PM involves lower surgical costs than mastectomy, post-PM radiotherapy may lead to higher total treatment cost for PM *vs* mastectomy.[22]

REFERENCES

1. Fisher B: A biological perspective of breast cancer: Contributions of the National Surgical Adjuvant Breast and Bowel Project clinical trials. *CA* 1991;41:97–111.
2. Fisher B, Anderson S, Fisher ER, et al: Significance of ipsilateral breast tumour recurrence after lumpectomy. *Lancet* 1991;338:327–31.
3. NIH Consensus Conference: Treatment of early-stage breast cancer. *JAMA* 1991;265:391–5.
4. Coleman A, Baker A: Stat bite: Trends in breast cancer surgery. *J Natl Cancer Inst* 1991;83:1607.
5. Fentiman IS, Mansel RE: The axilla: Not a no-go zone. *Lancet* 1991;1:221–3.
6. Blichert-Toft M, Axelsson CK, and Graversen HP: Axillary dissection in breast cancer. *Lancet* 1991;1:988.
7. Ries LAG, Hankey BF, and Edwards BK: *Cancer Statistics Review*, 1973–87. NIH Publication No. 90–2789. U.S. Public Health service, Bethesda, Md, 1990.
8. Ragland KE, Selvin S, and Merrill DW: Black-white differences in stage-specific cancer survival: Analysis of seven selected sites. *Am J Epidemiol* 1991;133:672–82.
9. Wilson RE, Donegan WL, Mettlin C, et al: The 1982 national survey of carcinoma of the breast in the United States by the American College of Surgeons. *Surg Gynecol Obstet* 1984;159:309–18.
10. Shambaugh E: Stat bite: Accuracy of SEER data. *J Natl Cancer Inst* 1991;83:1369.
11. Kjaergaard J, Blichert-Toft M, Andersen JA, et al: Probability of false negative nodal staging in conjunction with partial axillary dissection in breast cancer. *Br J Surg* 1985;72:365–7.
12. Kinne DW: The surgical management of primary breast cancer. *CA* 1991;41:71–83, 1991.
13. Chassin MR: Quality of care: Time to act. *JAMA* 1991;266:3472–3.
14. Harlan L: Current and new patterns of care studies. Presented at SEER Abstractor/Coder Workshop, Rockville, Md, 18 May 1992.
15. Osteen RT: Breast cancer. In Steele GD Jr, Winchester DP, Menck HR, et al. *National Cancer Data Base: Annual Review of Patient Care*, 1992. Atlanta GA: American Cancer Society, 1992, pp. 29–39.
16. Yancik R, Ries LG, Yates JW: Breast cancer in aging women: A populaton-based study of contrasts in stage, surgery, and survival. *Cancer* 1989;63:976–81.
17. Lazovich D, White E, Thomas DB, et al: Underutilization of breast-conserving surgery and radiation therapy among women with stage I or II breast cancer. *JAMA* 1991;266:3433–8.
18. Satariano ER, Swanson GM, and Moll PP: Nonclinical factors associated with surgery received for treatment of early stage breast cancer. *Am J Public Health* 1992;82:195–8.
19. Ganz PA, Schag AC, Lee JJ, et al: Breast conservation *vs* mastectomy: Is there a difference in psychological adjustment or quality of life in the year after surgery? *Cancer* 1992;69:1729–38.
20. Lasry JC, Margolese RG: Fear of recurrence, breast-conserving surgery, and the trade-off hypothesis. *Cancer* 1992;69:2111–5.
21. Lazovich D, Moe RE, Thomas D, et al: (Reply to letter to the editor) Breast-conserving surgery and radiation therapy: Are they underused? *JAMA* 1992;268:869–70.
22. Munoz F, Shamash F, Friedman M, et al: Lumpectomy *vs* mastectomy: The costs of breast preservation for cancer. *Arch Surg* 1986;121:1297–1301.

The Maternity Home for Adolescents: A Concept from the Past Fulfilling a Contemporary Need

ELAINE E. YORDAN, M.D. AND ROBERT A. YORDAN, M.D.

ABSTRACT—Maternity homes endure as an important means of imparting total care to some of today's homeless pregnant girls. This description of one such program is intended to serve as a model for the modern maternity residence for teens. Homelessness is a major complication of adolescent pregnancy. We believe that a supervised group home can be utilized to provide comprehensive care for homeless pregnant young women. We compiled data over the past six years (1986-91) from a residential facility for pregnant teenagers and their newborns, where the principal coauthor serves as voluntary medical director. During this period, 285 homeless adolescent females were admitted to the facility; their average age was 15.75 years, and 50% were white, 37% were African-American, and 13% were latino. All of the residents who were assigned private bedrooms, received prenatal care, accredited on-site schooling, family-life education, social services, dietary counseling, day-care services, and meals.

The average length of stay was four months. The average birth weight was 6.62 pounds. Sixteen percent of the newborns were placed for adoption. Data from 1987 through 1989 showed that 74% of the girls had either attained their educational or vocational objectives or were pursuing those goals at the time of discharge. Achievements included high school graduation, job training, and college attendance.

We conclude that this type of residential environment provides a wide range of resources beneficial to the homeless pregnant teen and her baby.

Introduction

As we approach a new century, those of us concerned with adolescent health care issues are focusing our attention and efforts toward the dual problems of homeless youth and adolescent pregnancy.[1,2] The contemporary image of a 13-year-old girl living on the streets of San Francisco, at high risk for disease and engaging in survival sex, is actually over 100 years old.[3] In the 19th century, charitable organizations began to establish group homes that provided specialized services for homeless, pregnant young women. The Catholic Church, the Salvation Army, and the Florence Crittenton movement were all credited with providing shelter, medical care, and social services to pregnant street youth and their babies throughout the United States beginning in the mid 1880s.[4]

Founded by the Sisters of Mercy in 1914, Saint Agnes Family Center in West Hartford is the only maternity residence for teens in Connecticut. Over the decades, multiple social and financial factors have been responsible for changes in the size and scope of the services provided at Saint Agnes Family Center. Its current functions are directed to the complex problems of today's homeless, pregnant teenagers and their newborns.

Our purpose is to describe the program at Saint Agnes Family Center, examine its effect, and suggest how it may serve as a model for others.

Materials and Methods

Saint Agnes Home, Incorporated, is an independent, nonprofit organization under the auspices of the Archdiocese of Hartford. It is licensed as a maternity home by the State of Connecticut Department of Health Services, the town of West Hartford, and the State of Connecticut Department of Children and Youth Services. The current site, a 10,000 square foot steel and concrete, air conditioned two-story structure, was originally built in 1968 as a convent. This location includes administrative offices, classroom space, recreational area, housekeeping and laundry facilities, private bedrooms, nursery, kit-

---

ELAINE E. YORDAN, M.D., Departments of Pediatrics, Saint Francis Hospital and Medical Center, Hartford, Connecticut, University of Connecticut School of Medicine, Farmington. ROBERT A. YORDAN, M.D., Department of Obstetrics and Gynecology, University of Connecticut School of Medicine, Farmington.

Reprints and Correspondence: Elaine E. Yordan, M.D., Department of Pediatrics, Section of Adolescent Medicine, Saint Francis Hospital and Medical Center, 114 Woodland Street, Hartford, Connecticut 06105.

Table 1

	1986	*1987*	*1988*	*1989*	*1990*	*1991*
Total number of girls admitted to the program	72	69	50	36	28	24
Number of antepartum admissions	45	46	34	25	23	19
Number of postpartum admissions	27	23	16	11	5	5
Average age of antepartum admissions	15.6	15.5	16.3	15.8	16.0	15.3
Number of babies born	36	35	27	15	12	16
Number of babies placed for adoption	6	8	5	4	0	0
Average length of stay (months):						
Antepartum at St. Agnes Home	2.8	2.8	2.4	2.5		
Postpartum at Lourdes Family Center	2.9	2.0	6.2	8.2		
Combined program at St. Agnes Family Center					5.6	6.1

chen, dining, and living rooms. It has a capacity for 16 residents and their babies. The staffing comprises 13 full-time and 12 part-time salaried positions. There are several additional health-care professionals who provide services and expertise on a voluntary basis. These include a physician, psychologist, and nurse practitioner. Members of the general public and civic organizations also volunteer their time and efforts to this program. Funding is provided by entitlement programs, private contributions, corporate donations, foundation grants, and the Archdiocese of Hartford. Admission to Saint Agnes Family Center is strictly voluntary and limited to young women under the age of 18 years without regard to race or religious affiliation. Admission is not available to young women currently dependent on illicit drugs or alcohol, or to those who constitute a physical danger to themselves or others. Those living at Saint Agnes Family Center are either pregnant teenagers or teen mothers and their babies. The maximum length of stay is two years.

There are several goals pursued at Saint Agnes Family Center: to provide the accepted standard of prenatal, intrapartum, postpartum, and newborn medical care; to teach sound personal health practices, childcare, and the skills necessary for independent living in the community; and to develop and implement a vocational or educational plan that will assist each young woman to reach her potential.

Pediatric, adolescent medicine, and obstetrical services are provided through the clinical facilities at Saint Francis Hospital and Medical Center in Hartford. A health education curriculum is taught by a full-time registered nurse. Topics include substance abuse, human sexuality, sexually transmitted diseases, interpersonal relationships, domestic violence, and refusal skills. A program of individual and group instruction in parenting and childcare is also taught by the registered nurse. A certified childbirth educator conducts classes on prepared childbirth. She works with the teens individually when necessary to insure full comprehension of the subject matter. Two registered dietitians are available for nutritional counseling for the teens and their babies. They also act as consultants for kitchen and food service planning. A full-time cook prepares all the meals. The vocational and educational components of the program begins with an individual assessment of each student's grade level. The West Hartford public school system provides five full-time teachers to Saint Agnes Family Center. Instruction is given to the pregnant teens on site as part of the home-bound program. After delivery, on-

Table 2

Racial Distribution at St. Agnes Family Center

	1986	*1987*	*1988*	*1989*	*1990*	*1991*
White	54.1%	46.5%	49.1%	41.6%	64.2%	41.6%
African-American	36.1%	38.3%	33.8%	41.6%	25.0%	50.0%
Latino	9.7%	15.0%	16.9%	16.6%	11.7%	8.3%

Table 3

Complications of pregnancy diagnosed among the 42 adolescent females admitted to St. Agnes Home, 1990 and 1991

Abnormal cervical cytology	2
Cervical *Neiseria gonorrheae* infection	1
Cervical *Chlamydia trachomatis* infection	5
Hepatitis A infection	1
Pyelonephritis	2
Cystitis	2
Sickle cell trait	1
Thalassemia minor	1
Hyperemesis gravidarum	1
Oligohydramnios	1
Pregnancy induced hypertension	2
Fetal distress	2
Preterm labor	2
Premature delivery	2
Post dates pregnancy	1

Table 4

Results of Educational and Vocational Development Program (EVDP) for 1987–89 for the 50 resident teenage mothers participating in the program.

20 (40%)	Had attained their EDVP objectives before their date of discharge.
17 (34%)	Were participating in EDVP at the time of discharge.
3 (6%)	Were registered to begin EDVP at time of discharge.
8 (16%)	Were in the referral or planning process for EDVP at the time of discharge.
1 (2%)	Had failed EDVP.
1 (2%)	Did not remain at the residence long enough to develop an EDVP plan.

site day-care is provided for the newborn to allow the teen mother to attend classes at the district public high school.

There are three social workers on the full-time staff. Key parts of each resident's care include a social work intake evaluation, identification of issues to be addressed, and formation of plans to meet specific needs.

The daily routines at Saint Agnes Family Center emphasize the importance of personal responsibility. This is achieved by a behavior modification system designed to encourage responsible and cooperative behavior by rewarding each resident for her own social growth and development.

Discharge and aftercare planning are part of the intake process. Each discharge plan is individualized according to the teen's needs and situation. Some of the alternatives available include returning home, foster placement, or independent living.

We performed a retrospective review of the annual statistics generated since 1986 (the year in which the principal author became medical director). From 1987 to 1989, the program was conducted at two sites, one antepartum and one postpartum. For financial reasons these were consolidated to one site in 1990. This change reduced the number of teens who could be admitted to the program by approximately one-half.

Results

From 1986 through 1991 there were 285 adolescent females admitted to the program. During this time, there were 192 antepartum admissions and 141 babies delivered (Table 1).

The average age of the pregnant teen at the time of admission was 15.75 years. The average length of stay for the pregnant girls admitted to the program was four months. The number of infants placed for adoption was 23 (16.3%). The racial distribution of the residents was 50% white, 37% African-American, and 13% latino (Table 2). Child protective services were responsible for referring 90% of the total admissions. The remainder came from hospitals (5%), local teen-parent programs (4%), and private families (1%).

During 1990 and 1991 there were 42 pregnant adolescent females admitted to the program. There were nine cesarean sections (21%) and two premature births (4.7%). The average birthweight during this time was 6.62 pounds with a range from 4 to 8.5 pounds. A variety of complications were diagnosed among these patients (Table 3). The median APGAR score was 8/9.

A special fund for education and vocational development was provided by a foundation grant for the three years from 1987 to 1990. There were 50 postpartum teens who participated in this program. Data showed that this investment yielded significant benefits (Table 4).

The financial cost of conducting this program involves employee salaries, supplies, building maintenance, consultant's fees, and administrative expenses (Table 5).

Discussion

Many communities are fortunate to have clinic-based adolescent prenatal programs, organized teen-parent programs, and educational systems that mainstream pregnant teens. The one simple but crucial constant upon which all such programs depend is that the pregnant teen has an intact homelife. What happens to the pregnant teen from a dysfunctional and disorganized family? Who will take care of the girl who gets pregnant and is no longer welcome in her parent's home? What refuge is there for the teen in a foster home who gets pregnant and is forced to leave? Where do you go if you are a pregnant teen living in a youth shelter? What kind of support system is available for the pregnant girl who has been adjudicated delinquent? We believe that a maternity home for adolescents can provide some of the solutions to these profound problems.

Over the years, the services provided by Saint Agnes Family Center have been modified to give better care and to insure the program's survival. Prior to 1983 Saint Agnes Home was strictly for prenatal patients. After delivery, the teen mothers and their babies had to leave. In 1983 a second facility, Lourdes Family Center, was established for teen mothers and their babies. Admission

Table 5

Annual budget of St. Agnes Family Center

	1989	*1990*	*1991*	*1992*
Revenue	$802,560.00	$770,945.00	$779,747.00	$759,580.00*
Expenses	$812,288.00	$795,370.00	$684,595.00	

* projected amount

was available not only to postpartum teens from Saint Agnes Home but also to teen mothers and their infants who had been delivered elsewhere. The two facilities had a combined capacity sufficient for 25 adolescent females.

In 1990 Lourdes Family Center was closed for financial reasons and the two programs were combined at one smaller site. This change is reflected by a decrease in the total number of girls admitted to the program. The number of antepartum admissions has decreased because of fewer available spaces. In addition, the State of Connecticut Department of Children and Youth Services currently tends to refer pregnant teens to less expensive sources of care whenever possible.

Although the number of girls served has decreased, the typical girl referred for admission comes from an increasingly disturbed background. Previously, Saint Agnes Family Center had numerous restrictive rules of daily living that prompted many teens to drop out of the program. Recently, liberalized policies have been implemented in an effort to motivate more girls to stay. It is now easier to earn privileges such as having visitors and leaving for the weekend. Yet, with these positive changes have come concerns that easier access to the negative influences outside Saint Agnes Family Center will result in exposure to illicit drugs, alcohol, and sexually transmitted diseases.

Although the average age of the girls at Saint Agnes Family Center is 15.75 years, since 1991 there have been three 13-year-old girls admitted to the program. Aside from the obvious problems, the younger teens are faced with some unique difficulties. As the maximal length of stay permitted at Saint Agnes Family Center is two years, new living arrangements will need to be found when they are just 15 years old.

Efforts have been made to provide special educational and vocational training for these girls. The goal has been to keep the girls in the program as long as possible, increasing their self-esteem through short-term accomplishments in vocational training and job placement.

The policy of the Saint Agnes Family Center is to discuss the issue of adoption during group counseling sessions. It is presented as one of the several available options. There were no babies placed for adoption in 1990 and 1991. In previous years, some teens included adoption as part of a long-range plan to reorganize their lives and complete an education. Recently, faced with strong peer pressure from girls in the program, those considering adoption have rejected this option.

In 1988 Connecticut reported 88 births for girls under the age of 15 years.[5] In the state 18% of high school students who entered ninth grade in 1984 did not graduate in 1988.[6] Connecticut has the third highest rate of babies born to unmarried teens in the nation.[7] The combination of teen motherhood, limited education, and no job skills precludes economic self-sufficiency for many young mothers.

At Saint Agnes Family Center, residents are given medical support, counseling, respect, guidance, and vocational and educational training. They are, quite simply, given a chance.

Acknowledgement

The authors wish to thank Mary Keegan, CISW, ACSW, and Penny Kollmeyer, MPA, for their assistance in the preparation of this manuscript and for their tireless work on behalf of the young women at Saint Agnes Family Center.

REFERENCES

1. Ad Hoc Committee on Reproductive Health: Society for Adolescent Medicine position paper on reproductive health care for adolescents. *J Adolesc Health* 1991;12: 649-61.
2. Pennbridge J, MacKenzie R, Swofford A: Risk profile of homeless pregnant adolescents and youth. *J Adolesc Health* 1991; 12:534-8.
3. Fielding E: Our rescue work. *War Cry* (Pacific Coast Division) 1888 April 15;2.
4. Niles G: Adoption agences, orphanages and maternity homes: An historical directory. Garden City, New York, Phileas Deigh Corporation, 1981, pp 279-86.
5. State of Connecticut, Department of Health Services: One *Hundred and Forty-first Registration Report for 1988*. Addendum, 1990;22-35.
6. Child Welfare League of America: Connecticut's children deserve more. *The Connecticut Priority* 1990; Fall.
7. Children's Defense Fund: The state of America's children, 1991. Washington, D.C., 1991, p 157.

On Death and Dying

STEWART P. SEIGLE, M.D.

ABSTRACT—Over a six-month period from February to August, 1992, one Hospice team cared for 134 patients who died. The terminal course of each patient was evaluated in terms of pain control, fear of death, and denial of death. Results were tabulated and conclusions were drawn.

Introduction

THIS was a cooperative study done by the South Team of Hospice of Southwest Florida. It was undertaken to see if we could offer a choice to people who were terminally ill that was better than suicide or assisted suicide. The terminal course and death of each of 134 patients who were seen by the team and died between February and August of 1992 were discussed and evaluated. At weekly death reviews, observations of the following were asked of each home health aid, nurse and social service counselor.

1. Control of pain: poor, fair, or good?
2. Fear of death: yes? no?
3. A state of denial—
    Patient: yes? no?
    Family: yes? no?

These observations and ratings were evaluated and recorded in relation to:

1. When the patient was first seen.
2. At the time of death.
3. Length of time with Hospice.

For many patients, comments amplifying the answers were recorded.

Possible reasons for suicide:

1. An easy solution?
2. Fear of pain?
3. Fear of dependency?
4. Fear of debility?
5. Depression?

The Role of Hospice

1. To provide physical and emotional support for both patient and family.
2. To alleviate pain.
3. To try to cope with role playing, denial, and fear.
4. To facilitate pastoral counseling when this is desired.
5. To provide volunteers to lighten family burdens.
6. To promote an easy and dignified death.

Diagnoses

During the five-month period, there were 134 patients who died; 50 women and 84 men. There were 38 different diagnoses listed as the cause of death. The three most common conditions were:

Cancer of the lung	37
Cancer of the colon	14
Cancer of the prostate	11

Other causes were found in decreasing numbers until finally there were 18 conditions with only a single death attributed to each one (Table 1).

Pain Control

Among the 134 patients, we had no case with intractable pain. There were 41 with inadequate pain control when first seen, and eight at the time of death. In general, a decision by the patient or the care giver not to use adequate medication was the cause for persistent pain in these eight patients (Table 2).

Patterns of Denial

At the outset, we found that 19 patients and 22 families denied the imminence of death. At the end, these numbers were 11 and 12, respectively. There were eight instances where the patient was in denial and the family not, and five when the family was in denial and the patient was not. There were three situations where both patient and family denied that death was imminent. We were uncertain about

---

STEWART P. SEIGLE, M.D., Volunteer, Hospice of Southwest Florida.

the feelings of the patient in five instances because of a short time with Hospice or because the patient was comatose when first seen. There was one family about which we could not be sure.

Table 1

Diagnosis

Male *84*
Female *50*
Total *134*

Diagnosis	*Total*	*Male*	*Female*
Cancer of colon	14	11	3
Cancer of prostate	11	11	
Cancer of pancreas	7	5	2
ASHD	6	3	3
Cancer of breast	5	1	4
Cancer of ovary	5		5
Cancer of bladder	5	4	1
CVA	4	1	3
Hypernephroma	3	2	1
Multiple myeloma	3	2	1
Cancer of rectum	3	3	
Renal failure	2	1	1
Malignant melanoma	2	1	1
ALS	2	2	
Parkinson's disease	2	2	
Cancer of liver	2		2
Pulmonary fibrosis	2	1	1
Acoustic neuroma	1		1
Acute myelogenous leukemia	1		1
Alzheimer's disease	1	1	
Anemia	1		1
Aneurysm and osteoporsis	1		1
Cancer head-face-neck	1	1	
Cancer of larynx	1	1	
Cancer of skin	1	1	
Cancer of parotid	1	1	
Cancer of pleura	1		1
Cancer of thyroid	1	1	
Cancer of tongue	1	1	
Cancer unknown	1		1
COPD	1	1	
Hepatitis B cirrhosis	1	1	
Hodgkins disease	1		1
HTLV	1	1	
Multiple sclerosis	1		1
Progressive bulbar palsy	1	1	
Total	134	84	50

		Cancer of lung
Male Deaths	63%	Male 62%
Female Deaths	37%	Female 38%

(See Table 2 on following page)

Fear of Death

Fifteen patients were afraid of dying when we first saw them and 12 were still fearful at the time of death. Of the three who changed, two were never in denial and the third was not at the time of death. Of the 12 who remained fearful four were never in denial, two were no longer in denial at the end, and six were always in denial. Of the families, four were in denial at first and only one at the end. Thus, of the 12 who died in fear, six still believed that they were not going to die.

Discussion

Terminal care was provided for patients with a wide variety of malignant and nonmalignant diseases. Of our patients 62% were male and 38% female. The most common diagnosis was cancer of the lung, and, of the 37 people who died of this disease, 62% were male and 38% female.

The fact that there were patients in denial when families were not, and families in denial when patients were not, suggests that in some instances role playing was present. Fear of death was associated with denial of death only about half the time, but for all patients in denial at the end death is final and their troubles are over. However, when families are in denial anger will interfere with normal grieving. It may be that denial is the only way these people can cope with fatal disease, but after death occurs this mechanism is no longer functional. These are the ones most in need of bereavement counseling even though they often reject it.

Among our patients, depression was present at times and it often responded to medication. Debility was minimized by the use of hospital beds, commodes, walkers, or other aids. Dependency was accepted gracefully by many, but some struggled to maintain control to the end. Fear of pain and lack of pain control was not a factor leading our patients to consider suicide, nor were there any suicides in the group.

Through the cooperative work of the Hospice team and the caring efforts of many volunteers, our patients were able to end their lives in an acceptable and dignified manner.

Conclusion

1. Hospice care does offer a viable and acceptable alternative to the choice of suicide as the best way to cope with terminal disease.
2. We should continue to consider how well we have handled the problems of fear, pain, and denial each time we close out a patient record.
3. The team approach of Hospice makes the care of terminally ill patients not only possible, but rewarding.

Table 2

Pain Control

Patients	134
Male	84
Female	50

Inadequate Pain Control First 41 Last 8

Summary of the Eight Not Well Controlled

Time with Hospice	*Age*	*Sex*	*Comment*	*Diagnosis*
6 weeks	47	M	Fearful of death and in denial, wife not in denial	Cancer of stomach
6 1/2 weeks	71	M	Patient and family in denial; medications poorly given	Cancer of prostate
22 weeks	59	F	Patient would not take medication; no one in denial	Cancer of ovary
4 days	81	M	Family hesitant about medication and Hospice, no denial	Cancer of lung
12 weeks	64	M	Patient chose to have pain; he and family in denial until the last week	Cancer of prostate
16 weeks	72	M	Pain not a problem save for a terminal myocardial infarction with death	ASHD; CHF
10 days	79	M	Had just come home from hospital—an angry person and wife did not give him his medication	Cancer of colon
4 weeks	35	M	Patient wanted to be alert and in charge; did not take adequate medication	Cancer of skin and Spina bifida

Be Part of the Excitement!

HEALTHCARE EXPO '93

Sponsored By CHA The Connecticut Hospital Association and Affiliates

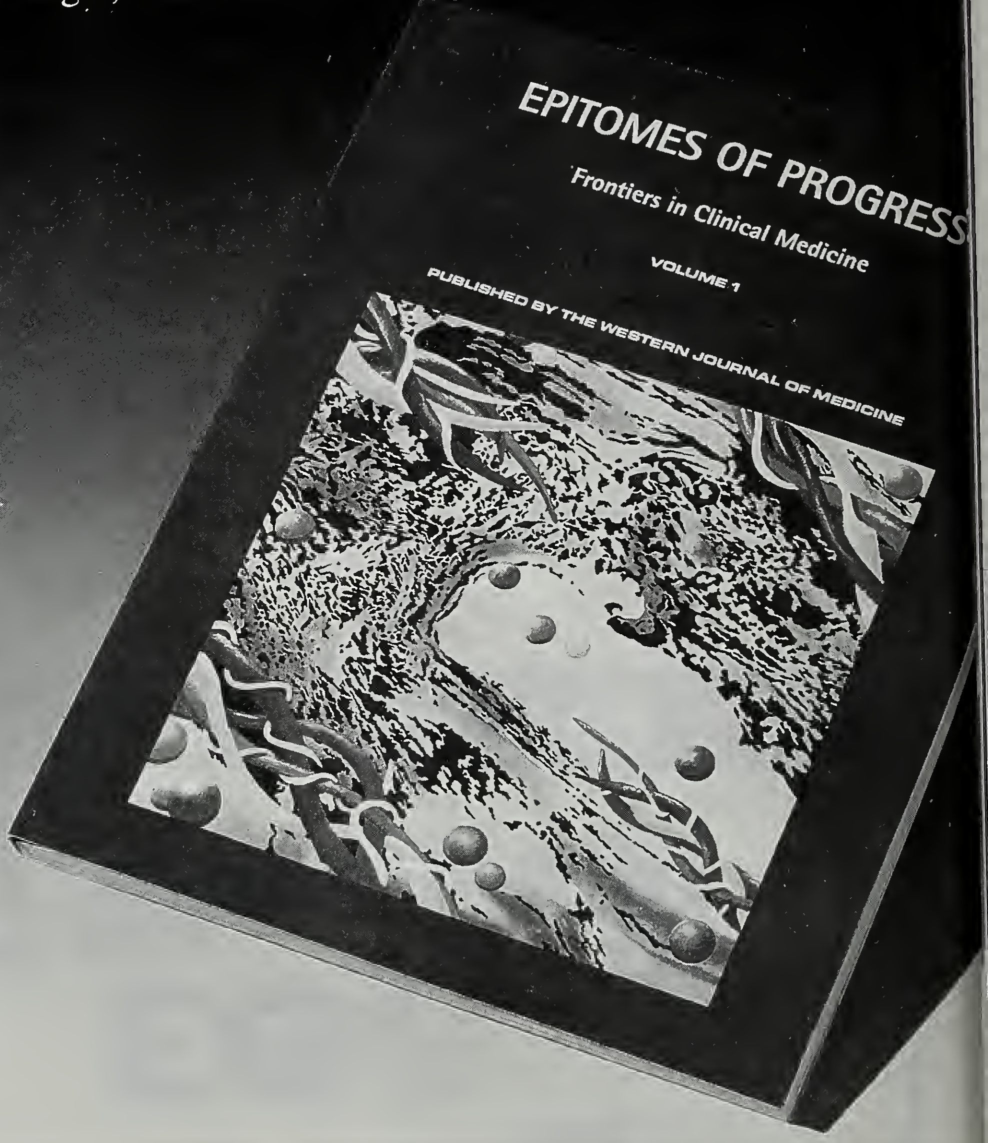
SAVE THE DATES: **APRIL 21 & 22, 1993**
HARTFORD CIVIC CENTER

- Over 285 exhibitors offering the most recent advances in products and services in the Healthcare Industry
- Travel giveaway
- **Special Physician Program** sponsored by The Connecticut State Medical Society
- Free Admission for all healthcare professionals

For More Information Call Robert Donnell Productions at 203-233-3654 • 800-243-9774

epitome

i-ˈpit-ə-mē\ *n* [L, fr. Gk *epitomē*, fr. *epitemnein* to cut short, fr. *epi-* + *temnein* to cut — more at TOME] (1520) **1 a** : a summary of a written work **b** : a brief presentation or statement of something **2** : a typical or ideal example: EMBODIMENT (the British monarchy itself is the ~ of tradition — Richard Joseph) **3** : Plural — *Epit-o-mes* — A regular FEATURE of *The Western Journal of Medicine*, now available in a single, bound volume.


EPITOMES OF PROGRESS
Frontiers in Clinical Medicine
VOLUME 1
PUBLISHED BY THE WESTERN JOURNAL OF MEDICINE

Each set of **EPITOMES** is a concise and clear review of the important advances in a medical specialty, chosen and written by experts in that specialty. Now, for the first time, a special bound volume of **EPITOMES** is available. For only $19.95 you can have all the latest advances in 24 different specialties in one attractively designed volume. An excellent reference for your library. A useful gift. Hurry! Supplies are limited. Order a copy today.

YES, SEND A SPECIAL BOUND EDITION OF **EPITOMES** TO:

The Western Journal of Medicine

☐ Yes, send me ____ copy(ies) of the Epitomes book at $19.95 each.

☐ I want to receive Epitomes every month. Please enter my one-year subscription to *WJM* for only $40.00.

For additional information, call (415) 882-5179.
Please mail this coupon with enclosed payment to:
Circulation Department
P.O. Box 7602, San Francisco, California 94120-7602

Name ____________________
Street ____________________
City ____________________
State ____________ Zip ____________
☐ Check enclosed   Charge my: ☐ VISA ☐ MasterCard
Card Number ____________________
Expiration Date ____________________
Signature ____________________

Important Advances in Clinical Medicine

Chest Diseases

The Council on Scientific Affairs of the California Medical Association presents the following inventory of items of progress in chest diseases. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome, and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, researchers, or scholars to stay abreast of these items of progress in chest diseases that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Chest Diseases of the California Medical Association, and the summaries were prepared under its direction.

Occupational Asthma

WHILE the overall prevalence of occupational asthma is unknown, it is estimated that from 5% to 15% of all cases of asthma in workers is attributable to workplace exposures. Occupational asthma can be defined as a variable airflow limitation caused by a specific agent in the workplace. Variable and sometimes chronic airflow limitation can result from sensitization to, pharmacologic effects of, recurrent inflammation from, or direct irritation by agents in the workplace. The treatment of occupational asthma may differ depending on its cause. The cornerstone of management is early diagnosis and identification of the offending agent, but diagnosis is not easy and often involves the investigation of exposure conditions at the workplace combined with an objective confirmation of exposure-related changes in lung function.

It is a common misconception that occupational asthma caused by a worker's sensitivity to a specific agent—for example, toluene diisocyanate or laboratory animal protein—resolves after the worker is removed from further exposure to that agent. Although occupational asthma often resolves with this intervention, the results of several longitudinal studies indicate that most persons continue to have some degree of persistent asthma years after removal from exposure. Factors associated with a more favorable result are a short duration of symptoms before diagnosis and relatively normal lung function and mild, nonspecific airway hyperresponsiveness at diagnosis. Early diagnosis and removal from exposure may prevent or lessen persistent asthma and the resulting impairment or disability.

Workers who continue in jobs that expose them to an agent to which they are sensitized tend to have increased respiratory symptoms, a progressive loss of ventilatory function, and increased airway responsiveness. This deterioration may occur despite suppressing symptoms with medication or reducing the intensity of exposure by using respiratory protective gear. Several deaths have been reported among workers with isocyanate-induced asthma who continued to work in jobs that involved exposure to that agent.

Some patients with occupational asthma cannot change jobs because of financial constraints. In such cases, vigorous efforts to reduce exposure to the lowest possible level are required. Product substitution, enclosure of specific work processes, and improved ventilation are preferable to the use of respirators, which are cumbersome, uncomfortable, and may leak. If respirators are to be used, however, patients must be medically certified, fit-tested, and properly trained to wear them. Careful monitoring can help assess the effectiveness of

---

Reprinted from *The Western Journal of Medicine,* August 1992.

Reprint requests to Division of Scientific and Educational Activities, California Medical Association, P.O. Box 7690, San Francisco, California 94120-7690

interventions to reduce exposure. Serial measurements of peak expiratory flow rates can document any recurrent subclinical bronchospasm, and serial methacholine challenge can assess whether the level of airway responsiveness is increasing. Patients with evidence of bronchospasm or an accelerated annual rate of decline in ventilatory function should be strongly encouraged to change jobs.

Drug therapy for occupational asthma is the same as that for persons with nonoccupational asthma, but treatment with drugs cannot substitute for eliminating exposure.

John R. Balmes M.D.
San Francisco, California

REFERENCES

Balmes JR: Surveillance for occupational asthma. *Occup Med* 1991; 6:101-10.

Chan-Yeung M: Occupational asthma. *Chest* 1990; 98 (suppl):148S-61S.

Fabbri LM, Danieli D, Crescioli S, et al: Fatal asthma in a subject sensitized to toluene diisocyanate. *Am Rev Respir Dis* 1988; 137: 1494-8.

Hudson P, Cartier A, Pineau L, et al: Follow-up of occupational asthma caused by crab and various agents. *J Allergy Clin Immunol* 1985; 76:682-8.

Antibiotics for Pneumonia

ACUTE pulmonary infection continues to cause substantial morbidity and mortality even in immunocompetent patients. Although a sputum Gram's stain may be immediately available and, if of good quality and showing a predominant organism, may help focus therapy, rapid and definitive identification of the specific etiologic agent(s) responsible for a pneumonia is often difficult. For these reasons, empiric antibiotic therapy is usually instituted concurrent with attempts at diagnosis. Epidemiologic factors are helpful in choosing a reasonable regimen.

A recent multicenter study of patients admitted for community-acquired pneumonia to a private community hospital, a university hospital, and a Department of Veterans Affairs hospital reported the four most common etiologic agents to be *Streptococcus pneumoniae* (15.3%), *Haemophilus influenzae* (10.9%), *Legionella* species (6.7%), and *Chlamydia pneumoniae* (6.1%). These findings strongly support including erythromycin in the empiric antibiotic treatment of community-acquired pneumonia. Two new macrolide antibiotics, clarithromycin and azithromycin, may be better tolerated and as effective as erythromycin in treating legionella, chlamydial, and mycoplasmal infections, although they are sevenfold more expensive.

Other pathogens responsible for community-acquired pneumonia are more common in specific patients. *Staphylococcus aureus is* observed in nursing home residents and following influenza infection. *Moraxella (Branhamella) catarrhalis* causes pneumonia in older patients and those with obstructive lung disease. Erythromycin generally is effective as the initial empiric therapy for infection with these organisms . More specific therapy for *M catarrhalis* and *H influenzae* includes the combination drugs amoxicillin and potassium clavulanate, ampicillin and sulbactam sodium, trimethoprim and sulfamethoxazole, ciprofloxacin, or a third-generation cephalosporin. Aerobic gram-negative bacilli are seen more commonly in older patients, those who use alcohol, and those with obstructive lung disease. Ciprofloxacin is not an ideal choice for empiric coverage of pneumonia because of marginal activity against *S pneumoniae.* In a severely ill or immunocompromised patient with community-acquired pneumonia of unknown origin, gram-negative bacilli must be covered.

Most pneumonias that develop in immunocompromised and hospital patients are caused by aerobic gram-negative bacilli. The most common agents are those of the family Enterobacteriaceae—*Klebsiella pneumoniae, Escherichia coli, Enterobacter* species, and *Serratia marcescens*—and *Pseudomonas* species. Several third-generation cephalosporins have excellent activity against these organisms and are good choices for these patients. Current evidence continues to support using a semisynthetic penicillin (piperacillin sodium) or a third-generation cephalosporin (ceftazidime) in synergistic combination with an aminoglycoside to treat pseudomonad infections. *S aureus* may be seen in the nosocomial setting and should be covered with initial therapy or if gram-positive cocci are observed in pulmonary secretions. Many third-generation cephalosporins such as ceftizoxime provide *S aureus* coverage. If methicillin-resistant *S aureus* is present in the hospital, vancomycin hydrochloride should be used in initial empiric regimens.

Aspiration pneumonia occurring in the community may be treated effectively with penicillin, but the increasing incidence of resistant *Bacteroides* species makes clindamycin hydrochloride a more reliable choice. Patients in hospital have high rates of colonization of the oropharynx and gastrointestinal tract with gram-negative bacilli. Antibiotic therapy for aspiration in the hospital must cover the usual nosocomial organisms as well as anaerobic pathogens; monotherapy with a third-generation cephalosporin such as ceftizoxime is reasonable empiric coverage.

Pneumonia caused by *Pneumocystis carinii*, viruses, and fungi *(Aspergillus* species, *Candida* species) may also develop in immunocompromised patients, requiring agents not typically included in initial empiric regimens. A recent randomized trial has shown that therapy with broad-spectrum antibiotics plus trimethoprim-sulfamethoxazole and erythromycin was as effective as and caused fewer complications than open-lung biopsy in cancer patients with diffuse pulmonary infiltrates. It is accepted practice to add empiric antifungal therapy for neutropenic patients who do not respond to treatment with antibacterial agents after several days.

In all settings, when a specific etiologic agent is identified, antimicrobial therapy should be narrowed to avoid superinfection and the selection of drug-resistant bacteria. With regard to preventing nosocomial pneumonia in critically ill patients, a recent well-controlled study has shown that selective decontamination of the digestive tract does not improve survival in intubated patients receiving mechanical ventilation in intensive care units.

Michael S. Bernstein, M.D.
San Francisco, California

REFERENCES

Browne MI, Potter D, Gress J, et al: A randomized trial of open lung biopsy versus empiric antimicrobial therapy in cancer patients with diffuse pulmonary infiltrates. *J Clin Oncol* 1990; 8:222-9.

Fang GD, Fine M, Orloff J, et al: New and emerging etiologies for community-acquired pneumonia with implications for therapy—A prospective multicenter study of 359 cases. *Medicine* (Baltimore) 1990; 69:307-16.

Gastinne H, Wolff M, Delatour F, Faurisson F, Chevret S: A controlled trial in intensive care units of selective decontamination of the digestive tract with nonabsorbable antibiotics. *N Engl J Med* 1992; 326:594-9.

Neu HC: New macrolide antibiotics: Azithromycin and clarithromycin. *Ann Intern Med* 1992; 116:517-9.

Diagnosis of Pulmonary Embolism

THE actual incidence and natural history of pulmonary embolism are unknown. Perhaps 30% of patients are symptomatic and have substantial morbidity and mortality. Diagnosis is essential because treatment is thought to reduce mortality. Symptoms and signs are nonspecific, and examination of leg veins is helpful only infrequently.

Pulmonary angiography is the gold standard for diagnosis but is invasive, often inconvenient, and, in some institutions, done too infrequently to approach the accuracy reported from major centers. Noninvasive screening includes radionuclide lung scanning and examination of the leg veins by impedance plethysmography and duplex ultrasonography. The positive predictive value of any test, however, is a function of the clinical probability that the condition exists.

Lung scanning is an excellent screening test. A normal scan predicts with almost 100% probability that pulmonary embolism has not occurred. Contrariwise, a high-probability scan is associated with thromboembolism in 50% to 90% of patients. The probability of a true-positive is a function of the clinical science probability. Unfortunately, a high-probability scan is seen in only 10% to 15% of patients and is, therefore, nonsensitive. The dilemma is how to proceed when the scan is intermediate, indeterminate, or of low probability in a patient with at least a moderate risk for pulmonary embolism.

A useful approach is to examine the leg veins with impedance plethysmography or duplex ultrasonography. The sensitivity and specificity of these tests in patients with symptoms of deep-vein thrombosis is reported to be in excess of 90%. In asymptomatic patients after a hip operation, impedance plethysmography was found to have an unacceptably low sensitivity of 24%. Another series found that 30% of patients with angiographically demonstrated pulmonary emboli had normal leg venograms.

If a patient has a moderate likelihood of pulmonary embolism, an intermediate- or low-probability scan, and negative leg vein studies, do we proceed with pulmonary angiography or do we observe? A report from Canada suggests that if leg vein studies remain negative in this cohort, treatment can be safely withheld. These investigators do emphasize the need for serial leg vein studies and also call for confirmation of their findings.

Anthony M. Cosentino, M.D.
San Francisco, California

REFERENCES

Agnelli G, Cosmi B, Ranucci V, et al: Impedance plethysmography in the diagnosis of asymptomatic deep vein thrombosis in hip surgery. *Arch Intern Med* 1991; 151:2167-71.

Hull RD, Raskob GE, Coates G, Panju AA, Gill GJ: A new noninvasive management strategy for patients with suspected pulmonary embolism. *Arch Intern Med* 1989; 149:2549-55.

Pedersen OM, Aslaksen A, Vik-Mo H, Bassoe AM: Compression ultrasonography in hospitalized patients with suspected deep venous thrombosis. *Arch Intern Med* 1991; 151:1217-2220.

The PIOPED Investigators: Value of the ventilation/perfusion scan in acute pulmonary embolism. *JAMA* 1990; 263:2753-9.

Thrombolytic Treatment of Pulmonary Embolism

OVER the past several years, there has been renewed interest in the use of thrombolytic therapy for treating venous thromboembolic disease. This is a consequence of both the availability of new thrombolytic agents and the interest in clot dissolution prompted by advances in treating coronary thrombolysis.

At present, the Food and Drug Administration has approved three thrombolytic agents for the treatment of pulmonary embolism: streptokinase, urokinase, and recombinant tissue plasminogen activator (rt-PA). Only streptokinase has been approved for treating deep venous thrombosis. All of these agents lower fibrinogen, plasminogen, and α_2-antiplasmin levels and generate a systemic lytic state. Fibrin selectivity has not yet proved to be of clinical importance nor has it been associated with lower rates of bleeding complications. Cost does differentiate among the several available thrombolytic agents. Although total costs vary with the duration of therapy, the total amount of the thrombolytic agent administered, the varied requirements for monitoring and blood work that different patients and protocols necessitate, and the occurrence and cost of complications, streptokinase is the least expensive of the available agents.

According to the standard protocols for treating venous thromboembolism, streptokinase and urokinase are administered by continuous intravenous infusion for periods of 12 to 72 hours, whereas tissue plasminogen activator is given as a 100-mg dose over two hours. Despite these recommendations, it is clear the optimal dosing regimens and duration of thrombolytic treatment have not yet been established. For continuous infusion therapy, setting the duration of therapy by the clock makes little sense; once thrombolytic treatment is initiated, it should be continued until either all clot amenable to thrombolysis has been dissolved or a complication of the therapy has been identified. One possible approach is to monitor the level of fibrin-degradation products (fibrin-split products), continuing thrombolytic treatment until levels are normal, indicating the cessation of notable clot lysis. For rt-PA, it is not clear if the short duration of therapy—based on the coronary thrombolysis model—is always sufficient to lyse the relatively large clot volume found in substantial pulmonary emboli and proximal deep venous thrombosis.

Current studies of the regimens for thrombolytic treatment of pulmonary emboli take into account the differences between the circulating half-lives of the thrombolytic agents (less than 25 minutes) and the duration of fibrinolytic activity within the thrombus (as long as six hours). Combining the duration of pharmacologic effects with the observation that an increased diffusion of thrombolytic agents into thrombus takes place when high circulating drug levels are present has led to the development of bolus-dosing protocols for treating pulmonary emboli with thrombolytic agents. Studies of animals and initial clinical trials with both urokinase and tissue plasminogen activator suggest that this approach may be more efficacious, with possibly less risk of sustained bleeding than the standard continuous infusion regimens.

Many investigators think that thrombolytic therapy should be used to treat any pulmonary embolus causing hemodynamic instability or respiratory compromise. More recently, some have proposed using echocardiography to evaluate large pulmonary emboli in stable patients; any evidence of right ventricular dysfunction or dilation is used as a rationale for treating with thrombolytic agents. This approach is currently being evaluated in a randomized trial.

A decade ago it was reported that the diffusing capacity of the lungs for carbon monoxide in patients treated with thrombolytic therapy was better preserved than in those treated with heparin. The explanation was that the pulmonary capillary blood volume (pulmonary microcirculation) is better preserved after thrombolytic therapy than after standard heparin anticoagulation. A preliminary report of follow-up hemodynamic examinations of these patients found that those treated with thrombolytic agents had lower mean pulmonary arterial pressures and pulmonary vascular resistance at rest and during exercise than did those who had been treated with heparin. The significance of the modest hemodynamic differences is unclear, however, and the question of whether the routine use of thrombolytic therapy for less substantial pulmonary emboli is appropriate to preserve the pulmonary microcirculation remains unanswered.

Harold I. Palevsky, M.D.
Thomas A. Raffin, M.D.
Stanford, California

REFERENCES

Agnelli G, Parise P: Bolus thrombolysis in venous thromboembolism. *Chest* 1992; 101:172S-82S.

Come PC: Echocardiographic evaluation of pulmonary embolism and its response to therapeutic interventions. *Chest* 1992; 101:151S-62S.

Goldhaber SZ: Thrombolysis in venous thromboembolism: An international perspective. *Chest* 1990; 97:176S-81S.

Palevsky HI, Fishman AP: Diagnosis and treatment of pulmonary embolism and deep venous thrombosis, *In* Fishman AP (Ed): *Update: Pulmonary Disease and Disorders*. New York, NY, McGraw-Hill, 1992, pp 451-64.

Lung-Assist Devices

TWO kinds of devices are used to assume the gas-exchange task of the lung. The first is an artificial lung, which is needed in part because of the lack of a suitable supply of healthy lungs for transplantation. The second is a lung-assist device to bridge the gap until lung transplantation can be accomplished. It is also used in patients with a severe abnormality in the gas-exchange process, as in the adult respiratory distress syndrome. Lung-assist devices may also help reduce intrathoracic pressure when patients are on mechanical ventilation, possibly resulting in decreased morbidity and mortality associated with the adult respiratory distress syndrome. The two approaches to lung-assist devices currently being evaluated are extracorporeal carbon dioxide removal and intravascular oxygenator.

In 1974 the National Institutes of Health funded a study on the efficacy of extracorporeal membrane oxygenation in the adult respiratory distress syndrome. The control group was treated with mechanical ventilation and positive end-expiratory pressure. The result in both groups was about 10% survival, leading to the conclusion that extracorporeal membrane oxygenation has no role in this disorder. In the early 1980s, neonatologists had better success in the treatment of neonatal respiratory distress syndrome, with an overall survival rate reaching 78%.

Several recent European reports have suggested that the survival rate for patients treated with extracorporeal membrane oxygenation who met the entry criteria of the original 1974 study was close to 50%, reviving interest in lung-assist devices. There have been several important improvements in the technology, such as thromboresistant coating of the surfaces used, improved efficiency of membranes, and better pumps to reduce hemolysis and platelet destruction. The major change was that extracorporeal membrane oxygenation was modified into extracorporeal CO_2 removal. In the former procedure, blood completely bypasses the heart and the lungs. This technique is designed to support the failing lungs and heart. In extracorporeal CO_2 removal, blood is drained from and returned to the vena cava after membrane gas exchange, therefore only supporting the lungs. Extracorporeal CO_2 removal also allows the use of low-frequency mechanical ventilation, low positive end-expiratory pressure, and continuous low-flow oxygen, but oxygen uptake continues to occur mainly in the lungs. The technique is labor intensive, requires capital equipment, and runs at high cost.

The intravascular oxygenator is an intracorporeal gas-exchange device placed in the vena cava. It is constructed with polypropylene hollow fibers coated with ultrathin biocompatible siloxane. All blood-contacting surfaces are also coated with covalently bonded heparin. Oxygen is sucked through the fibers, gas exchange occurs through the membranes, and blood is oxygenated intravenously. Placement and maintenance are relatively simple and inexpensive. Because of its relatively small surface area, its capacity is only a third to half of the gas exchange necessary to maintain aerobic metabolism.

Thus, extracorporeal membrane oxygenation is an acceptable technique in the management of neonates with respiratory distress syndrome. In adults, all three techniques are still experimental but have promise. The development of a totally artificial lung remains some time away.

David M. Gelmont, M.D.
South Pasadena, California

REFERENCES

Anderson HL III, Delius RE, Sinard JM, et al: Early experience with the adult extracorporeal membrane oxygenation in the modern era. *Ann Thorac Surg* 1992; 53:553-63.

Lally KP, Clark R, Schebdeman C, et al: Extracorporeal membrane oxygenation in the newborn. *Milit Med* 1990: 155:377-9.

Bronchiolitis Obliterans Organizing Pneumonia

THE term bronchiolitis obliterans organizing pneumonia (BOOP) was coined in 1985 to describe a lung disease characterized histologically by the presence of intraluminal fibroblastic proliferation. In Europe, BOOP is also known as cryptogenic organizing pneumonitis. It has recently been suggested that the disorder be renamed as cryptogenic intramural fibrosis of distal air spaces.

Bronchiolitis obliterans organizing pneumonia is divided into primary or idiopathic and secondary. The secondary BOOP may occur in association with certain infections—with *Legionella, Mycoplasma,* or *Adenovirus* species—the inhalation of toxic gases (nitrogen dioxide), graft-versus-host disease in bone marrow and heart-lung transplant recipients, and collagen vascular diseases. It is, however, the idiopathic or primary BOOP that enters in the differential diagnosis of diffuse interstitial lung disease.

Because of certain common clinical and physiologic abnormalities, BOOP is often misdiagnosed as idiopathic pulmonary fibrosis, sarcoidosis, pneumoconiosis, hypersensitivity pneumonitis, or chronic eosinophilic pneumonia. It is important that BOOP, because of its benign course and favorable response to corticosteroids, is not overlooked or misdiagnosed.

Idiopathic BOOP occurs mainly in adults, with a peak incidence in the sixth decade; women and men are affected equally. Most patients have a subacute illness with cough, dyspnea, fever, and fatigue of a few weeks' duration. The patient's sedimentation rate is often elevated. Although auscultation may reveal rales, in a third of patients the results of a pulmonary examination are normal. Hypoxemia occurs in almost all patients. Lung volumes are reduced, but airway obstruction is not a feature. Diffusing capacity is impaired. Chest roentgenograms show bilateral patchy airspace densities, commonly in peripheral areas, and many other radiographic patterns have also been reported. Honeycombing is rare. The conventional and high-resolution computed tomographic scan findings are nonspecific, but these new techniques are helpful in distinguishing BOOP from idiopathic pulmonary fibrosis and granulomatous lung disease. On histologic examination, the lungs display immature-appearing plugs in the respiratory bronchioles, alveolar ducts, and peribronchial alveolar spaces. The alveolar spaces also contain foamy cells reflecting the proximal airway obstruction. Alveolar septa are thickened and contain mononuclear cell infiltrate. The presence of intraluminal plugs of fibroblasts, however, distinguishes BOOP not only from idiopathic pulmonary fibrosis but also from other interstitial lung disorders.

When should a clinician think about a diagnosis of bronchiolitis obliterans organizing pneumonia? Internists and pulmonologists should consider including BOOP in the differential diagnosis of a subacute interstitial pneumonia, particularly when the patient has influenza-like features with cough, dyspnea, fever, malaise, and weight loss. A poor or unsatisfactory response to antibiotics is another feature favoring this diagnosis. A good transbronchial lung biopsy specimen in conjunction with a strongly suggestive clinical history and examination is in many cases adequate to establish the diagnosis. An open-lung biopsy is needed when a transbronchial biopsy fails to establish the diagnosis. This pneumonia has an excellent prognosis. It responds well to corticosteroids; almost two thirds of the patients in one study improved with the use of corticosteroids. Fewer than 5% of patients die of respiratory failure.

Idiopathic bronchiolitis obliterans organizing pneumonia was recently added to the long list of interstitial disorders. The disease has a benign course with an excellent response to corticosteroids, but we remain ignorant of its cause.

Om P. Sharma, M.D.
Los Angeles, California

REFERENCES

Bellomo R, Finlay M, McLaughlin P, Tai E: Clinical spectrum of cryptogenic organizing pneumonitis. *Thorax* 1991; 46:554-8.

Miyagawa Y, Nagata N, Shigematsu N: Clinicopathological study of migratory lung infiltrates. *Thorax* 1991; 46:233-8.

Muller NL, Staples CA, Miller RR: Bronchiolitis organizing pneumonia: CT features in 14 patients. *AJR* 1990; 154:983-7.

Advances in Pulmonary Biopsy Techniques

In the past decade, refinements in transthoracic and transbronchial fine-needle aspiration (FNA) techniques, the introduction of thoracoscopic biopsies, and the improvement in lesion imaging and specimen processing have led to a continued decline in the need for open lung biopsies.

The main indications now for percutaneous transthoracic FNA are the biopsy of pulmonary nodules and aspiration of infectious lesions. The diagnostic yield of conventional transbronchial forceps biopsy diminishes with the size of the lesion, whether infectious or neoplastic, so that lesions less than 2 cm to 3 cm are best approached by FNA. In immunocompromised patients, in whom early diagnosis is paramount, small subsegmental and peripheral lesions should undergo FNA under computed tomographic or fluoroscopic guidance because a positive result provides both a diagnosis and confirmation of invasion for otherwise potentially saprophytic organisms like fungi.

Transbronchial FNA by flexible bronchoscopy has the advantage of a lower risk of pneumothorax than percutaneous methods. Its 50% yield in peripheral lesions supplements conventional transbronchial forceps biopsy, washing, and brushing, so that using them together increases the overall yield to about 70%. Compared with transbronchial forceps biopsy, transbronchial FNA is a better technique for metastatic lesions because these tend not to be peribronchial in distribution and hence require deeper biopsies. In addition, about 50% to 60% of malignant mediastinal lymph nodes in paratracheal, subcranial, and aorticopulmonary sites can be sampled with transbronchial FNA guided by computed tomographic images. When successful in the evaluation of lung cancer, this method provides both a cytologic diagnosis and staging, without the need for mediastinoscopy. Because transbronchial FNA has only moderate sensitivity, a negative result does not rule out malignant adenopathy, and it should be noted that the method provides no information about whether disease has spread through the involved node's capsule, a known factor in prognostication. Other uses of transbronchial FNA include the biopsy of extrabronchial compressive lesions and superficially necrotic tumors that require deeper biopsies.

Newer molecular biology and immunohistologic tools have partially eliminated the need for the larger biopsies obtainable only by thoracotomy. Immunohistologic methods using antibodies directed against cell surface antigens can usually separate, for example, reactive lymphocytosis from lymphoma. Gene rearrangement techniques are so sensitive that even cytologic specimens obtained by bronchoalveolar lavage may be adequate for diagnosing certain lymphomas. A polymerase chain reaction assay for *Mycobacterium tuberculosis* holds similar promise for very small or even expectorated specimens in tuberculosis.

When large pleural or parenchymal specimens are required, diagnostic thoracoscopy is now an option. Under general anesthesia, a double-lumen tube is placed to permit collapse of the lung. A rigid thoracoscope is inserted by trocar for plain or video viewing. An operating probe or second thoracoscope, which can incorporate the use of therapeutic carbon dioxide or neodymium-yttrium-aluminum garnet lasers, is positioned separately to permit wedge excisional biopsy or even segmentectomy. In pleural diseases, thoracoscopy has proved invaluable in diagnosis, and in appropriate cases, therapeutic pleural debridement and even ablation of pleural blebs can be accomplished using these techniques.

Norman W. Rizk, M.D.
Stanford, California

REFERENCES

Shure D: Transbronchial biopsy and needle aspiration. *Chest* 1989; 95:1130-8.

Wakabayashi A: Expanded applications of diagnostic and therapeutic thoracoscopy. *J Thorac Cardiovasc Surg* 1991; 102:721-3.

Wang KP, Kelly SJ, Britt JE: Percutaneous needle aspiration biopsy of chest lesions—New instrument and new technique. *Chest* 1988; 93:993-7.

Lung Transplantation

HUMAN lung transplantation actually predated heart transplantation, beginning with a single-lung transplant done in 1963. Over the next 15 years, about 44 single-lung transplants were attempted worldwide. The results were uniformly dismal, however, and the procedure was abandoned. Studies of animals suggested that en bloc transplantation of the heart and both lungs provided better vasculature to the tracheobronchial tree, leading to the first successful human heart-lung transplant at Stanford University Medical Center in 1981. This procedure is successful for combined cardiopulmonary disease, particularly congenital heart disease and right ventricular failure associated with pulmonary hypertension. Combined heart-lung blocks are in limited supply, however, stimulating efforts to use lung-only grafts.

Single-lung transplantation was successfully reintroduced in 1983. Major principles advocated were selecting recipients with fibrotic disease only; wrapping the bronchial anastomosis with omentum; not giving preoperative corticosteroids; and avoiding administering maintenance corticosteroids in the early postoperative period. Over the past five years the original principles have been progressively modified: Single-lung transplantation is appropriate for patients with fibrotic disease, emphysema, and perhaps pulmonary hypertension; omental wrapping of the bronchial anastomosis is not required; patients may be successfully transplanted while receiving low dosages of corticosteroids; and instituting corticosteroid therapy immediately following transplantation may be acceptable.

Double-lung transplantation was introduced in 1986. The original procedure involved a low tracheal anastomosis and was technically flawed, with a high rate of tracheal dehiscence. It has now been replaced with bilateral bronchial anastomoses with improved results.

The results of lung and heart-lung transplantation continue to improve with one- and five-year survival rates approaching 75% and 50%, respectively. Candidates for transplantation should have a life expectancy of less than two to three years without transplantation and be otherwise healthy. Most programs set age limits for transplantation, such as age 60 for single lung, age 50 for double lung, and age 45 for heart-lung transplantation. Rejection monitoring requires frequent assessment of pulmonary function along with bronchoscopy and transbronchial biopsy. The long-term immunosuppression regimen consists of cyclosporine, azathioprine, and prednisone.

Long-term immunosuppression places transplant patients at risk for various unusual infections. In addition, obliterative bronchiolitis, possibly a manifestation of chronic lung allograft rejection, develops in some recipients and is the major cause of late graft failure. Frequent monitoring of pulmonary function and transbronchial biopsy may allow early detection of this process, but obliterative bronchiolitis progresses inexorably once it is established, despite augmentation of the immunosuppressive regimen.

The major limitation of lung or heart-lung transplantation continues to be an insufficient number of suitable donors, inequities in transplantation funding, and late graft failure from obliterative bronchiolitis. Nonetheless, lung and heart-lung transplantation have become accepted therapeutic options for many patients with heretofore fatal pulmonary disorders.

E. Clinton Lawrence, M.D.
James Theodore, M.D.
Vaughn A. Starnes, M.D.
Stanford, California

REFERENCES

Calhoon JH, Grover FL, Gibbons WJ, et al: Single lung transplantation: Alternative indications and technique. *J Thorac Cardiovasc Surg* 1991; 101:816-25.

Lawrencc EC: Diagnosis and management of lung allograft rejection. *Clin Chest Med* 1990; 11:269-78.

Theodore J, Starnes VA, Lewiston NJ: Obliterative bronchiolitis. *Clin Chest Med* 1990; 11:309-21.

Monoclonal Therapy for Bacteremia and Sepsis

GRAM-NEGATIVE bacteremia occurs in 300,000 to 500,000 patients each year. The mortality ranges from 10% to 80% and is highly dependent on a patient's underlying condition and concurrent disease processes. Gram-negative bacteremia is associated with organ failure, which increases mortality, especially if more than one organ is affected. Studies have been done that predict in which septic patient organ failure will develop and, therefore, who will have the highest morbidity and mortality. These predictors have not yet been used clinically, however.

Antiendotoxin antibodies have been used to treat bacteremic patients and patients who appear "septic" despite negative blood cultures. The infusion of endotoxin recapitulates a sepsis-like illness in animals; endotoxin levels are not always detectable in patients with sepsis or even in those with bacteremia, however. Nonetheless, the infusion of polyclonal antiserum to lipid A in patients with gram-negative bacteremia can reduce mortality from 39% to 22%. Patients in septic shock also have had a reduction in mortality, but patients with septic neutropenia do not appear to benefit. The antiserum has never been marketed because of lot-to-lot variation and concern regarding blood products.

Two monoclonal antibodies against endotoxin have recently been developed: HA-1A is a "humanized" immunoglobulin M (IgM) antibody, and E5 is a murine IgM antibody. In clinical studies, the HA-1A was found not to reduce morbidity and mortality in patients with bacteremia or sepsis. In a subset of 200 patients with gram-negative bacteremia with or without shock, mortality was reduced, and there was a trend for a decrease in organ failure in patients with bacteremia treated with the antibody. More patients in the placebo group had renal failure and hepatic failure and had *Pseudomonas aeruginosa* infections, however, leaving some question as to whether the treatment and placebo groups were comparable.

In a study of patients treated with E5, mortality was not reduced, but in a subset of patients who had gram-negative bacteremia but who were not hypotensive, mortality was reduced and there was a trend for less organ failure. A second trial used E5 to treat normotensive septic patients who, conversely, did not show a reduction in mortality. There did seem to be a benefit to the patients who received E5 in terms of a resolution of organ failure, however.

These therapies are expensive. Independent investigators estimated a cost of $24,100 per year of life saved with optimal drug use. They suggested a worst-case scenario, however, of a cost of $1,227,600 per year of life saved. A prudent suggestion would be to await further studies of these agents in selected bacteremic patients, including neutropenic and other immunosuppressed patients, to see if these agents are efficacious in all patients with bacteremia. Also, a comparison study of the two antibodies would clarify some of these issues. Other costly agents will be available shortly, such as antitumor necrosis factor and interleukin receptor antagonists; physicians must be critical of the cost-benefit of these drugs for a condition that has an extremely variable prognosis.

Joe Guglielmo, PharmD
Jeanine P. Wiener-Kronish, M.D.
San Francisco, California

REFERENCES

Greenman RL, Schein RMH, Martin MA, et al: A controlled clinical trial of E5 murine monoclonal IgM antibody to endotoxin in the treatment of gram-negative sepsis. *JAMA* 1991; 266:1097-102.

Schulman KA, Glick HA, Rubin H, Eisenberg JM: Cost-effectiveness of HA-1A monoclonal antibody for gram-negative sepsis—Economic assessment of a new therapeutic agent. *JAMA* 1991; 266:3466-71.

Ziegler EJ, Fisher CJ Jr, Sprung CL, et al: Treatment of gram-negative bacteremia and septic shock with HA-1A human monoclonal antibody against endotoxin. *N Engl J Med* 1991; 324:429-36.

Conventional Treatment of Cystic Fibrosis

TWENTY years ago cystic fibrosis meant death by age 7; today the median survival age is 28. This remarkable progress has been accomplished through nutritional support, tracheobronchial care, and multidisciplinary therapy.

Nutritional support is focused on oral ingestion of pancreatic enzymes, high food intake, and vitamin and mineral supplements, particularly the lipid-soluble vitamins A, E, K, and sometimes D. The need for lipid-soluble vitamins is assessed directly by the measurement of vitamin A and vitamin E levels and indirectly by the measurement of prothrombin time and serum calcium levels for vitamins K and D. Acid-resistant pancreatic enzymes have contributed substantially to improved survival rates. The acid resistance allows the enzymes to pass undigested through the stomach into the duodenum. Food requirements for patients with cystic fibrosis can be 20% to 50% higher than normal for at least two recognizable reasons: Patients with cystic fibrosis have some malabsorption despite enzyme replacement, and their metabolic rate is high, probably because of bronchial inflammation and the increased work of breathing. Patients with advanced lung disease may have poor appetites. The use of a gastrostomy tube is gaining acceptance because nutrition status and longevity are correlated.

Tracheobronchial care is less well defined but generally consists of the use of inhaled and oral bronchodilators, antibiotics, and clearing mucus by postural drainage and percussion, sometimes augmented with mucolytics. An active exercise program is also useful. These elements are based on clinical experience and common sense but lack vigorous scientific validation, except that patients are living longer.

Antibiotics are used liberally in these patients. Increased sputum production, decreased appetite, and shortness of breath are enough to indicate a need for antibiotic therapy. Fever, leukocytosis, or a new infiltrate on a chest roentgenogram are even more imperative signals. Sedimentation rate and quantitative C reactive protein, measures of acute-phase reactants, are used by some as additional indicators for administering antibiotics. The typical antibiotic course is long—14 to 21 days. Some patients use antibiotics nearly continuously, sometimes administered by aerosol in those with substantial secretions. Antibiotic use is guided by sputum cultures: *Haemophilus influenzae, Staphylococcus aureus,* and, later in life, *Pseudomonas aeruginosa* are the usual pathogens. Many antibiotics have an accelerated halflife in these patients, and dosage has to be adjusted accordingly.

Postural drainage and percussion preceded by an inhaled B-agonist is traditionally recommended twice a day. Lung transplantation, with a 50% survival at two years, is considered for patients with end-stage lung disease.

Cystic fibrosis is a complex disease with numerous medical, psychiatric, and social consequences. For patients and their families, it means a lifetime of adjustments. Patients can be managed by individual physicians but should also be registered with a cystic fibrosis center for consultation and provision of otherwise unavailable services. Such centers are identified by the National Cystic Foundation (1-800-FIGHT CF) and in California by the State Genetically Handicapped Persons Program (916-654-0503) and California Children's Service (916-654-0499).

Bertrand J. Sharpiro, M.D.
Los Angeles, California

REFERENCES

Fifth Annual North American cystic Fibrosis Conference. *Pediatr Pulmonol* 1991; 6(suppl): 1-329.

Kopelman H: Cystic fibrosis: Gastrointestinal and nutritional aspects. *Thorax* 1991; 46:261-7.

Mouton JW, Kerrebijn KF: Antibacterial therapy in cystic fibrosis. *Med Clin North Am* 1990; 74:837-50.

Aerosolized Bronchodilators

BECAUSE of direct delivery to the site of action, inhaled bronchodilators are rapidly effective even in small quantities and, hence, cause a minimum of systemic side effects. Whether bronchodilators are inhaled through a nebulizer, through a metered-dose inhaler (MDI) with or without spacer, or through a dry powder inhaler (DPI), only about 10% to 20% of the inhaled drug traverses the major barrier of the upper airways and is available for deposition on potentially beneficial spots in lower airways. When selecting a form of aerosol administration, physicians should weigh the advantages and disadvantages of each type.

Nebulizers are expensive, labor intensive, and inconvenient compared with MDIs and DPIs. Unusual bronchodilators such as the anticholinergic glycopyrrolate can be delivered only by nebulizer. The waste in nebulizers is in the loss of drug in tubing and apparatus. Though nebulizers have been widely used for years, adjustment of the major elements of operation including fill volume, operating pressure and flow, and length of time of nebulization is not well established; hence, nebulizers are frequently not used well. Though nebulizers are as efficient as MDIs, amounts of medication used and delivered are much greater, and, hence, the possibility of side effects from overdosage is much greater.

Because of their convenience of size and the availability of most major bronchodilators, MDIs are widely used. Appropriate technique requires the coordination of canister activation with slow inhalation and breathholding. While most patients can learn this technique, some will require aids such as spacers. Because spacers are less convenient, alternatives such as breath-actuated devices may be useful.

Though some drugs are available in the dry powder inhaler format, its use is not common. Within several years, when freon propellants are no longer available, DPIs probably will be used more frequently. A possible advantage of the DPI is that it delivers a drug as efficiently as a nebulizer or MDI without requiring coordination between activation and inhalation. New DPIs deliver several doses of a drug without the inconvenience of reloading.

Archie F. Wilson, M.D., PhD.
Orange, California

REFERENCE

Zainudin BM, Biddiscombe M, Tolfree SEJ, Short M, Spiro SG: Comparison of bronchodilator responses and deposition patterns of salbutamol inhaled from a pressurized metered dose inhaler, as a dry powder, and as a nebulized solution. *Thorax* 1990 45:469-73.

Screening for Obstructive Sleep Apnea Using Pulse Oximetry

RECENT research has indicated that obstructive sleep apnea is much more common than generally appreciated. The reported prevalence is 1% to 5% in unselected populations and even greater in selected populations such as habitual snorers (30% to 50%). It is also serious, with an 8- to 11-year mortality of about 30%, affecting individuals through associated disease and society through sleep disruption and its effects on job performance and driving. At present, the recommended diagnostic tool is nocturnal polysomnography, but the expense, inconvenience, and lack of general availability of this test have deprived patients of the benefit of treatment.

The appropriate screening tool for obstructive sleep apnea remains to be defined. A patient's history may be highly suggestive, but only that, in 60% to 70%—snoring, observed apneas, and obesity, particularly truncal, are the most predictive. Examination will rarely be specific, even the direct observation of sleep. Pulmonary function tests are also not helpful; the sawtooth pattern of the flow-volume curve has been discredited as a marker. The non-neurophysiologic elements of the polysomnogram have also been suggested, but the absence of airflow, the sine qua non of obstructive sleep apnea, is difficult to record easily. The electrocardiogram often shows a characteristic, repetitive tachycardia-bradycardia, a consequence of the repetitive apneas, but the presence of an autonomic neuropathy will mask this.

Pulse oximetry has been shown to be highly specific (100%) but, using current accepted criteria for abnormal desaturations (less than or equal to 4%), is not sufficiently sensitive (60% to 70%). Two recent studies have highlighted this. At present, therefore, the screening of populations by visual analysis of pulse oximetry traces alone will lead to an underestimate of the prevalence of obstructive sleep apnea. In selected populations where clinical suspicion exists— sleepy snorers, for example—a negative or indeterminate pulse oximetry trace may not obviate the need for further study. Because oximetry is so specific, however, a positive study may allow the physician to introduce therapy such as nasal continuous airway pressure, with an abolition of symptoms being the criterion for adequate treatment.

An additional confounding problem is the presence of the periodic limb movement disorder in some patients, which can independently cause substantial sleep deprivation. The prevalence increases with age—5% at 30 to 50 years of age and 44% at 65 and older—and has been found in 5% to 18% of patients suspected of having obstructive sleep apnea.

Because of the lack of sensitivity of oximetry and the coexistence of other disorders causing sleep disruption, screening for obstructive sleep apnea using only oximetry will not obviate the need for further study in a symptomatic patient. The electroencephalogram may not be essential, but, in addition to oximetry and clinical assessment, a measurement of respiratory activity and of leg movements is probably needed in the evaluation for this disorder as further refinement of the interpretation of the oximetry tracing is undertaken.

Adrian J. Williams, M.D.
Myron Stein, M.D.
Los Angeles, California

REFERENCES

Douglas NJ, Thomas S, Jan MA: Clinical value of polysomnography. *Lancet* 1992; 339:347-50.

Stradling JR, Crosby JH: Predictors and prevalence of obstructive sleep apnoea and snoring in 1,001 middle aged men. *Thorax* 1991; 46:85-90.

Williams AJ, Stein M: Clinical value of polysomnography (Letter). *Lancet* 1992; 339:1113.

Williams AJ, Yu G, Santiago S, Stein M: Screening for sleep apnea using pulse oximetry and a clinical score. *Chest* 1991; 100:631-5.

ADVISORY PANEL TO THE SECTION ON CHEST DISEASES

Lowell E. Renz, M.D.
Advisory Panel Chair
CMA Council on Scientific Affairs Representative
Sacramento

Herbert M. Schub, M.D.
CMA Section Chair
Alameda

John L. Sherman, M.D.
CMA Section Secretary
Los Angeles

Michael L. Cohen, M.D.
CMA Section Assistant Secretary
Walnut Creek

Philip M. Gold, M.D.
Loma Linda University

Thomas A. Raffin, M.D.
Stanford University

Glen Lillington, M.D.
University of California, Davis

Archie Wilson, M.D.
University of California, Irvine

Daniel H. Simmons, M.D., Ph.D.
University of California
Los Angeles

Jack L. Clausen, M.D.
University of California
San Diego

Stephen C. Lazarus, M.D.
University of California
San Francisco

Om P. Sharma, M.D.
Section Editor
University of Southern California

Henry Gong, Jr, M.D.
American College of Chest Physicians
Los Angeles

Anthony M. Cosentino, M.D.
American College of Chest Physicians
San Francisco

Christopher J. Newth, M.D.
California Thoracic Society
Los Angeles

Thomas E. Addison, M.D.
California Thoracic Society
San Francisco

National CFIDS Expert to Speak in Connecticut

David S. Bell, M.D., FAAP, will be the guest speaker at Manchester Memorial Hospital in Manchester, Connecticut on 28 March 1993. His topic will be **Chronic Fatigue and Immune Dysfunction Syndrome: An Update**. The general session, open to the public, will be from 2:00 P.M. to 4:00 P.M. At 4:00 P.M., Dr. Bell will hold a special session for physicians and other health professionals only. This portion of the meeting will be an advanced question and answer session. Refreshments will be served during this medical reception. Those planning to attend the second session are asked to RSVP by **March 23, 1993** to Dr. Dana Wiseman of Glastonbury at 657-4800, or to Dr. Robert Weiss of West Hartford at 236-6155.

In 1985, Dr. Bell was one of the first to characterize CFIDS (also known as Chronic Epstein-Barr Virus, CFS, Myalgic Encephalomyelitis, Fibromyalgia, etc.) during a large cluster outbreak that occured in Lyndonville, New York. Since that time, he has maintained his stature as one of the foremost experts on the disease by conducting research, publishing numerous articles and a book, *The Disease of A Thousand Names*, and lecturing to physicians and patients across the country. Dr. Bell is an instructor in pediatrics, Harvard Medical School, Boston, Massachusetts, and the Department of Pediatrics, The Cambridge Hospital, Cambridge, Massachusetts. He is a member of the board of The CFIDS Association of America, and serves on its Scientific Advisory Committee.

The Connecticut CFIDS Association, Inc. is sponsoring this event.

Because One Size Doesn't Fit All...

New Humulin 50/50 is the tailor-made answer to individual patient needs. A unique combination of equal amounts of Regular human insulin and NPH human insulin, it will be useful in situations in which a greater initial insulin response is desirable for greater glycemic control.

Like Humulin 70/30,* new Humulin 50/50 offers the convenience and accuracy of a premix. And it can be used in conjunction with an existing 70/30 regimen.

New Humulin® 50/50

50% human insulin isophane suspension
50% human insulin injection
(recombinant DNA origin)

The Newest Option in Insulin Therapy

WARNING: Any change of insulin should be made cautiously and only under medical supervision.

* Humulin® 70/30 (70% human insulin isophane suspension, 30% human insulin injection [recombinant DNA origin]).

Lilly

Global Excellence in Diabetes Care
Eli Lilly and Company
Indianapolis, Indiana
46285

HI-7911-B-249343 © 1992, ELI LILLY AND COMPANY

Drugs for Pain

THE MEDICAL LETTER

THREE types of analgesic drugs are available in the USA: first, aspirin, other nonsteroidal anti-inflammatory drugs (NSAIDs), and acetaminophen; second, opioids; and third, some drugs not usually thought of as analgesics, which act as adjuvants when given with NSAIDs or opioids, or have analgesic activity of their own in some types of pain (American Pain Society, *Principles of Analgesic Use,* 3rd ed, Skokie, Illinois: American Pain Society, 1992).

NSAIDs AND ACETAMINOPHEN—With increasing doses, aspirin, acetaminophen, and the newer NSAIDs all reach a limit on their maximum analgesic effect. For aspirin and acetaminophen, the maximum analgesic effect usually occurs with doses between 650 and 1300 mg. The ceiling for the newer NSAIDs may be relatively higher, and the duration of analgesic effect may be longer than with aspirin or acetaminophen. With all of these drugs, patients evidently do not develop tolerance to their analgesic effects.

Aspirin—Controlled clinical trials have shown aspirin to be effective in most types of pain, including cancer pain. Inhibition of cyclooxygenase is probably the mechanism of aspirin's analgesic action and also causes some of its adverse effects. A single therapeutic dose irreversibly inhibits platelet function for the seven-day lifetime of the platelet, inhibiting hemostasis and prolonging bleeding time. A single dose of aspirin can also precipitate aspirin-sensitive asthma. Multiple doses can cause gastropathy and salicylism.

Acetaminophen—Acetaminophen is as effective as aspirin, similar in potency, and, in single analgesic doses, has the same time-effect curve. Acetaminophen is not effective in rheumatoid arthritis, but in acute tissue trauma it may have anti-inflammatory effects comparable to those of aspirin and other NSAIDs (P Skjelbred et al, Curr Ther Res, 35:377, 1984). Acetaminophen does not cause the adverse effects characteristic of aspirin. Overdosage can cause serious or fatal hepatic injury, but most patients, except alcoholics or some with active liver disease, can take 4 grams a day with no adverse effects.

Other NSAIDs—In single full doses, most of the NSAIDs listed in the table on page 88 are more effective analgesics than full doses of aspirin or acetaminophen, and some can equal or exceed the analgesic effect of oral narcotic combination products, or even injected opioids. Whether this is also true with repeated doses in chronic pain is less well established, and few controlled trials comparing the newer NSAIDs with each other have been published. Some patients may respond better to one NSAID rather than another (WT Beaver in KM Foley et al, eds, *Advances in Pain Research and Therapy,* Vol. 16, New York: Raven Press, 1990, page 109).

Combinations—Aspirin, acetaminophen, or NSAIDs can be given concurrently with opioids for an additive analgesic effect. Even when insufficient alone to control pain adequately, NSAIDs have an additive effect that permits use of lower dosage of opioids. There is no evidence that NSAIDs have additive effects when used with each other.

Adverse Effects of NSAIDs—The adverse effects of all NSAIDs are similar to those of aspirin. Except for the risk of precipitating asthma and anaphylactoid reactions in aspirin-sensitive patients, single or a few doses of the newer NSAIDs cause few adverse effects. Unlike aspirin, NSAIDs cause reversible inhibition of platelet aggregation; platelet function returns when most of the drug has been eliminated.

With chronic use, upper gastrointestinal bleeding, ul-

Reprinted with permission from *The Medical Letter,* Vol. 35, January 8, 1993.

ceration, and perforation can occur, sometimes without warning. Some reports suggest that ibuprofen, naproxen, and ketoprofen cause less gastrointestinal toxicity than aspirin or other NSAIDs, but fatal hemorrhage and perforation have occurred with all of these drugs.

NSAIDs decrease synthesis of renal vasodilator prostaglandins, decrease renal blood flow, cause fluid retention, and may cause renal failure in some patients; risk factors include old age, congestive heart failure, chronic renal insufficiency, ascites, intravascular volume depletion, and concurrent diuretic therapy. NSAIDs rarely also cause allergic interstitial nephritis and the nephrotic syndrome, both usually reversible on discontinuation of the drug.

Unlike opioids, NSAIDs have no potential for abuse. Physical dependence on these drugs has not been reported.

Ketorolac—Ketorolac is the first injectable NSAID available for analgesic use in the USA (Medical Letter, 32:79, 1990). Other parenteral NSAIDs have been marketed in other parts of the world, but adverse reactions at the site of injection have limited their usefulness. Intramuscular (IM) ketorolac is relatively nonirritating to tissues. In one controlled trial, 30 mg of ketorolac IM was as effective and longer acting in postoperative pain than 12 mg of morphine IM (DA O'Hara et al, Clin Pharmacol Ther, 41:556, 1987). After oral surgery, a single 10-mg oral dose of ketorolac was superior to 650 mg of aspirin, 600 mg of acetaminophen, or an acetaminophen/codeine combination, and at least as effective as 400 mg of ibuprofen in controlled trials (JA Forbes et al, Pharmacotherapy, 10:77S; 94S, 1990).

OPIOIDS—Although usually considered the drugs of choice for severe acute pain and chronic cancer pain, the effectiveness of opioids overlaps that of the NSAIDs. Oral codeine, its congeners, propoxyphene, and pentazocine in usual doses are no more effective when taken alone than aspirin or acetaminophen and are usually prescribed in combinations with these drugs to treat moderate or moderately severe pain. Morphine, hydromorphone, methadone, levorphanol, fentanyl, and large doses of oxycodone are used for the most severe pain (KM Foley, N Engl J Med, 313:84, 1985). Opioids produce analgesia by binding to various opioid receptors within, and possibly outside, the central nervous system; they are classified as full agonists, partial agonists, and mixed agonist-antagonists. Morphine and the other full agonists, unlike NSAIDs, have no ceiling on their analgesic effectiveness, except that imposed by adverse effects.

Choice of Opioid—While the parenteral full agonists listed in the table on page 89 differ in potency, they are all, with the exception of codeine, equally capable of relieving acute pain in the nontolerant patient if the dose is adjusted appropriately.

Morphine—Morphine is the standard of comparison for strong injectable analgesics. Given orally, it is well absorbed but extensively metabolized on the first pass through the liver and has a short duration of action. Sustained-release oral preparations have a longer duration of action. The drug can also be given rectally, sublingually, and by application to the buccal mucosa (KV Shepard and AW Bakst, Cleve Clin J Med, 57:48, 1990). Morphine has no greater dependence liability than equally effective doses of any full agonist.

Meperidine—Given intramuscularly, meperidine is irritating to tissues and short acting. Repeated doses lead to accumulation of a toxic metabolite with a 15- to 20-hour half-life, which can cause dysphoria, irritability, tremors, myoclonus and, occasionally, seizures, particularly with large repeated doses or in patients with decreased renal function. In patients taking monoamine oxidase inhibitors, meperidine can cause severe encephalopathy and death *(Medical Letter Handbook of Adverse Drug Interactions,* 1993, page 171).

Methadone and Levorphanol—Both of these drugs are sometimes used orally to relieve pain in patients with cancer. Since, however, the average plasma half-life of methadone is 24 hours and that of levorphanol is 12 to 16 hours, repeated doses of either drug can lead to accumulation and progressive central-nervous-system (CNS) depression, particularly in elderly or debilitated patients.

Partial Agonists and Mixed Agonist-Antagonists—The partial agonist buprenorphine is similar to the mixed agonist/antagonists pentazocine, butorphanol, nalbuphine, and dezocine. All of these drugs can precipitate withdrawal symptoms in patients physically dependent on full agonists. All are less likely to cause dependence than full agonists, but none is completely free of dependence liability. Only pentazocine is available for oral use and only in combination products.

New Developments—Patient-controlled intravenous (IV) analgesia may offer advantages over IM injections for some patients (Medical Letter, 31:104, 1989). Intraspinal opioids have been used to control postoperative and cancer pain with few adverse effects (Medical Letter, 27:92, 1985; PH Allaire et al, Mayo Clin Proc, 67:1031, Nov 1992). Fentanyl is now available in a transdermal formulation (Medical Letter, 34:97, 1992), and butorphanol is marketed as a nasal spray (TK Abboud et al, Acta Anaesthesiol Scand, 35:14,1991).

Tolerance to Opioids—Tolerance is common with chronic use of opioids; the patient first notices a shorter duration of analgesia and then an increase in pain. Tolerance can be delayed by giving nonopioid analgesics concurrently. Tolerance to most of the adverse effects of opioids, including respiratory and CNS depression, develops at least as rapidly as tolerance to the analgesic effect. Tolerance, therefore, can usually be surmounted and adequate analgesia restored by increasing the dose. Cross-tolerance exists among all of the full agonists, but it is not complete, and switching to another opioid, starting with half the equianalgesic dose, may be helpful.

Dependence—Patients who take strong opioids will develop physical dependence and abstinence symptoms if the drug is discontinued suddenly or an opioid antagonist is given. Physical dependence can be detected after a few days of therapy, but clinically significant dependence develops only after several weeks of chronic treatment with relatively large doses of morphine-like opioids. Patients who take opioids for acute pain or cancer pain rarely experience euphoria and even more rarely develop psychic dependence or addiction to the mood-altering effects of narcotics.

Adverse Effects—Sedation, grogginess, dizziness, nausea, vomiting, and constipation are the most common adverse effects of opioids; respiratory depression is the most serious. Sedation can be ameliorated by decreasing the dose and increasing the frequency of administration, by switching to opioids with a shorter half-life, and by giving small oral doses of dextroamphetamine or methylphenidate *(Ritalin,* and others) in the morning and early afternoon (E Bruera et al, Pain, 50:75, 1992). Tolerance usually develops rapidly to the sedative and emetic effect of these drugs, but not to constipation.

In patients with chronic obstructive pulmonary disease, cor pulmonale, decreased respiratory reserve, or preexisting respiratory depression, even usual doses of opioids may decrease respiratory drive and cause apnea. Patients without pulmonary disease who take opioids chronically are often tolerant to the respiratory depressant effect, but the addition of general anesthetics, phenothiazines, tranquilizers, sedative-hypnotics, tricyclic antidepressants, or other CNS depressants increases the risk.

Dosage—Opioid dose requirements may vary widely from one patient to another. For the nontolerant patient, the usual dose must frequently be increased to achieve adequate analgesia and occasionally decreased to avoid adverse effects. For most strong opioids, the dose should be increased until adverse effects occur before switching to another opioid. Once the optimal dose that will provide adequate analgesia, hopefully for at least four hours, has been established by titration, it should be given on a schedule, with the provision that the patient can refuse a dose if not in pain. "By-the-clock" administration of analgesics is much more effective than waiting for severe pain to return before giving the next dose and may decrease total dosage. An order for a supplementary opioid dose between regular doses, which may be the same or different from the regular medication, should be available as a backup for breakthrough pain.

ADJUVANT ANALGESICS—Tricyclic antidepressants, such as amitriptyline *(Elavil,* and others), in doses lower than those used to treat depression can relieve many types of neuropathic pain, including post-herpetic neuralgia and diabetic neuropathy (MB Max et al, N Engl J Med, 326:1250, 1992). The anticonvulsants **carbamazepine** *(Tegretol,* and others), **clonazepam** *(Klonopin),* or **phenytoin** *(Dilantin,* and others) can relieve lancinating pain from trigeminal neuralgia, post-herpetic neuralgia, glossopharyngeal neuralgia, and neuralgias arising from nerve trauma or infiltration with cancer (M Swerdlow, Clin Neuropharmacol, 7:51, 1984).

Caffeine, although not an analgesic when given alone, in doses of 65 to 200 mg has been shown in controlled trials to enhance the analgesic effect of acetaminophen, aspirin, or ibuprofen in headache, oral surgery pain, and postpartum pain (EM Laska et al, JAMA, 251:1711, 1984; JA Forbes et al, Clin Pharmacol Ther, 49:674, 1991). **Hydroxyzine** *(Vistaril,* and others) in doses of 50 to 100 mg IM adds significantly to the analgesic effect of opioids in postoperative and cancer pain while reducing the incidence of nausea and vomiting.

Corticosteroids—Through their anti-inflammatory effect, corticosteroids can produce analgesia in some patients with inflammatory diseases or tumor infiltration of nerves (KM Foley and C Macaluso in GM Aronoff, ed, *Evaluation and Treatment of Chronic Pain,* 2nd ed, Baltimore:Williams & Wilkins, 1992, page 340).

Acetaminophen and NSAID Analgesics

Drug	Usual single analgesic dose	Dose interval	Maximum daily dose	Comments
Acetaminophen *(Tylenol,* others)	PO: 500-1000 mg	q4-6h	4000 mg	As effective as aspirin; 1000 mg usually more effective than 650 mg; duration of action usually 4 hours
Salicylates				
Aspirin (many manufacturers)	PO: 500-1000 mg	q4-6h	4000 mg	Duration of action after single doses usually 4 hours; active metabolite accumulates with repeated doses
Choline magnesium trisalicylate *(Trilisate)*	PO: 1000-1500 mg	q8-12h	3000 mg	Effectiveness compared to aspirin not clear; onset of analgesia probably slower; less gastropathy and impairment of platelet function
Diflunisal *(Dolobid)*	PO: 1000 mg initial then 500 mg	q8-12h	1500 mg	500 mg superior to 650 mg of aspirin or acetaminophen, with slower onset and longer duration; comparable to acetaminophen/codeine combination
Other NSAIDs				
Etodolac *(Lodine)*	PO: 200-400 mg	q6-8h	1200 mg	Efficacy data limited; 200 mg comparable and 400 mg possibly superior to 650 mg of aspirin
Fenoprofen *(Nalfon)*	PO: 200 mg	q4-6h	1200 mg	Comparable to aspirin; contraindicated with impaired renal function
Flurbiprofen *(Ansaid)*	PO: 50-100 mg	q6-8h	300 mg	Analgesic indication not FDA-approved; 50 mg superior to 650 mg of aspirin with longer duration of action; 100 mg superior to acetaminophen/codeine combination
Ibuprofen *(Motrin, Rufen, Advil, Nuprin,* others)	PO: 200-400 mg	q4-8h	2400 mg	200 mg equal to 650 mg of aspirin and acetaminophen; 400 mg superior with longer duration; 400 mg comparable to acetaminophen/codeine combination.
Ketoprofen *(Orudis)*	PO: 25-75 mg	q4-8h	300 mg	25 mg comparable to ibuprofen 400 mg and superior to 650 mg of aspirin; 50 mg superior to acetaminophen/codeine combination
Ketorolac *(Toradol)*	IM: 60 mg initial then 30 mg	q6h	120 mg	Comparable to 12 mg IM morphine or 100 mg IM meperidine with longer duration; 150 mg can be given first day; use should be limited to 5 days
	PO: 10 mg	q6h	40 mg	10 mg superior to aspirin or acetaminophen or acetaminophen/codeine combination and comparable to ibuprofen 400 mg; for short term use
Meclofenamate *(Meclomen)*	PO: 50-100 mg	q4-8h	400 mg	Comparable to aspirin; approved for dysmenorrhea
Mefenamic acid *(Ponstel)*	PO: 500 mg initial then 250 mg	q6h	1250 mg	Comparable to aspirin but more effective in dysmenorrhea; duration of use not to exceed 1 week
Naproxen *(Naprosyn)*	PO: 500 mg initial then 250 mg	q6-8h	1250 mg	250 mg probably comparable to 650 mg of aspirin with slower onset and longer duration; 500 mg superior to 650 mg of aspirin
Naproxen sodium *(Anaprox)*	PO: 550 mg initial then 275 mg	q6-8h	1375 mg	275 mg comparable to 650 mg of aspirin with slower onset and longer duration; 550 mg superior to 650 mg of aspirin with comparable onset

Opiod Analgesics

Drug	IM dose[1]	IM Duration (hours)[2]	Starting oral dose[3]	Comments
Agonists related to morphine				
Morphine *(Duramorph,* and others)	10 mg	3-6	20-60 mg	Available as 8-12h sustained release tablets *(MS-Contin, Oramorph-SR)*
Hydromorphone *(Dilaudid)*	1.3 mg	3-5	4-8 mg	Available as high potency injectable *(Dilaudid-HP)* and as suppository
Oxymorphone *(Numorphan)*	1.1 mg	3-5	—	Available as suppository
Heroin	4 mg	3-5	—	Adverse effects and dependence liability same as morphine
Agonists related to codeine				
Codeine	130 mg	3-5	30-60 mg	60 mg PO equivalent to 650 mg of aspirin or acetaminophen; usually used orally in combinations with these drugs; dependence liability < morphine
Dihydrocodeine *(Synalgos-DC)*	—	—	32 mg	Available in combination with aspirin; dependence liability probably < morphine
Hydrocodone *(Vicodin,* others)	—	—	5 mg	10 mg PO equivalent to codeine 80 mg PO; available in combination with acetaminophen or aspirin
Oxycodone *(Roxicodone,* others)	—	—	5 mg	10 mg PO equivalent to codeine 90 mg PO; available in combination with acetaminophen or aspirin
Synthetic opioid agonists				
Meperidine *(Demerol,* others)	80 mg	2-4	50 mg	Irritating to tissues IM; toxic metabolite with long half-life causes CNS excitation and convulsions
Fentanyl *(Sublimaze, Duragesic)*	0.1 mg	1-2	—	Used in anesthesia; transdermal patch for chronic pain releases fentanyl over 72 hours
Methadone *(Dolophine,* others)	10 mg	4-6	10-20 mg	Risk of CNS depression with repeated use; used in narcotic dependence
Propoxyphene HCI *(Darvon,* others)	—	—	65 mg	65 mg of HCI or 100 mg of napsylate PO equivalent to codeine
Propoxyphene napsylate *(Darvon-N)*	—	—	100 mg	32 mg PO; available in combinations with acetaminophen or aspirin; toxic metabolite with long half-life causes convulsions and cardiotoxicity; dependence liability < morphine
Levorphanol *(Levo-Dromoran)*	1.5-2.5 mg	4-6	2-4 mg	Risk of CNS depression with repeated use
Partial agonists and mixed agonist/antagonist				
Buprenorphine *(Buprenex)*	0.4 mg	4-6	—	Partial agonist; virtually no psychotomimetic effects; sublingual preparation effective
Pentazocine *(Talwin,* others)	60 mg	2-4	50 mg	Mixed agonist/antagonist; 50 mg PO equivalent to codeine 60 mg PO; very irritating to tissues; psychotomimetic effects
Butorphanol *(Stadol, Stadol NS)*	1.5-2.5 mg	3-6	—	Mixed agonist/antagonist; nasal spray produces analgesia comparable to IM injection
Nalbuphine *(Nubain)*	12 mg	3-6	—	Mixed agonist/antagonist; less psychotomimetic effects than pentazocine
Dezocine *(Dalgan)*	10 mg	3-6	—	Mixed agonist/antagonist

---

1. Equivalent to 10 mg of IM morphine
2. With an optimal dose established by titration
3. These drugs are usually given q4-6h, but the duration of action varies with different doses and different patients, and the schedule should be established by titration.

WHO SAYS YOU CAN'T JUGGLE IT ALL ?

ProMed says you can. As a medical provider you strive to keep your patients healthy. As a businessman you aim to run an organized and efficient office. At ProMed, we can provide you with a medical office system to meet your most basic business needs — or we can provide you the advantages of a multi-user, multi-tasking operating system. And, as your practice grows, so can your ProMed office system to meet your changing needs.

With ProMed, dedicated service and comprehensive training are only the beginning:

- ☑ Faster and more accurate electronic claims submission
- ☑ Reduced paperwork
- ☑ Detailed financial and clinical management reporting
- ☑ Complete installation and support services

As the leading provider of medical office systems in Connecticut, ProMed saves time and money for more than 1,000 of the state's medical professionals.

You take care of your patients, we'll take care of the rest. For more information, call Bob Seekamp at ProMed: 1-800-922-3385.

ProMed
SYSTEMS INCORPORATED
221 Whitney Avenue
New Haven, Connecticut 06511

An Historical Review of Rupture of the Heart

MARTIN DUKE, M.D.

In 1649 William Harvey noted the autopsy findings of a patient with spontaneous rupture of the heart. Additional reports by Nicholls, Morgagni, Hodgson, Quain, Vulpian, and others describing the dramatic clinical and pathological features of cardiac rupture appeared during the following 250 years. These reports are cited in order to document and trace an increasing awareness that acute myocardial infarction caused by obstructive coronary artery disease was the most frequent cause of this catastrophic event. Frequently these older reports speak for themselves without elaboration. At other times, they will be cautiously interpreted in the context of present-day understanding of this disease.

Cardiology Service, Manchester Memorial Hospital, Manchester.

But his flaw'd heart,
Alack! too weak the conflict to support;
'Twixt two extremes of passion, joy and grief,
Burst smilingly.
William Shakespeare
King Lear, Act V

Cardiac rupture of the free wall of the ventricle is one of the most devastating events in medical practice. Early descriptions of such cases during the 17th, 18th, and 19th centuries gradually began to note that rupture of the ventricle occurred primarily in the context of underlying coronary artery obstruction and acute myocardial infarction. By quoting from such references, this paper will present a historical perspective of cardiac rupture through the words of contemporary authors of that era. When appropriate, these will be discussed in light of current terminologies and concepts.

An Early Case Report

In 1649 William Harvey (1578-1657), best remembered for his work on the circulation of blood, commented on the clinical history and postmortem findings of a patient with a rent in the wall of the left ventricle, perhaps the earliest known description of cardiac rupture.[1]

> A noble knight, Sir Robert Darcy, an ancestor of that celebrated physician and most learned man, my very dear friend Dr. Argent, when he had reached to about the middle period of life, made frequent complaint of a certain distressing pain in the chest, especially in the night season; so that dreading at one time syncope, at another suffocation in his attacks he led an unquiet and anxious life. He tried many remedies in vain, having had the advice of almost every medical man. The disease going on from bad to worse, he by and by became cachectic and dropsical, and finally, grievously distressed, he died in one of his paroxysms. In the body of this gentleman, at the inspection of which there were present Dr. Argent, then president of the College of Physicians, and Dr. Gorge, a distinguished theologian and preacher, who was pastor of the parish, we found the wall of the left ventricle of the heart ruptured, having a rent in it of size sufficient to admit any of my fingers, although the wall itself appeared sufficiently thick and strong; this laceration had apparently been caused by an impediment to the passage of the blood from the left ventricle into the arteries (p. 127).

The symptoms of this patient suggest in part the syndrome that in later years William Heberden (1710-1801) would call angina pectoris.[2] Harvey made no specific mention of the coronary arteries, and terminology that might have suggested a myocardial infarction—eg, localized softening, scar, ulcer—was not used. The possible presence of long-standing heart failure resulting in cardiac cachexia, and of hypertrophy of the left ventricle caused by hypertension or even aortic stenosis, are also suggested in this patient. Further discussion would, however, be speculative without additional information.

The 18th Century: From King George II to Morgagni

In a report of particular interest because of the individual in whom the disease occurred, Frank Nicholls (1699-1778), a distinguished medical practitioner of this period and physician to the king, described the clinical and autopsy findings of George II of England, ruler from

---

MARTIN DUKE, M.D., Cardiovasular Diseases, Internal Medicine, Editorial Board of Conn Med.

Reprinted by permission of the publisher from Cardiovascular Pathology 1992; 1(3):237-7. Copyright ©1992 by Elsevier Science Publishing Col., Inc.

Address for reprints: Martin Duke, M.D., PO Box 967, Manchester, CT 06045-0967.

1727 to 1760:[3]

> ... his Majesty was found fallen on the ground, speechless and motionless.... He appeared to have just come from his necessary-stool, and as if going to open his escritoir....
>
> Upon opening the chest ... [and] examining the heart, its pericardium was found distended, with a quantity of coagulated blood, nearly sufficient to fill a pint cup; and, upon removing this blood, a round orifice appeared in the middle of the upper side of the right ventricle of the heart, large enough to admit the extremity of the little finger. Through this orifice, all the blood brought to the right ventricle had been discharged into the cavity of the pericardium; and, by that extravasted blood, confined between the heart and pericardium, the whole heart was very soon necessarily so compressed, as to prevent any blood contained in the veins from being forced into the auricles; which, therefore, with the ventricles, were found absolutely void of blood, either in a fluid or coagulated state.
>
> As, therefore, no blood could be transmitted through the heart, from the instant that the extravasation was completed, so the heart could deliver none to the brain; and, in consequence, all the animal and vital motions, as they depend on the circulation of the blood through the brain, must necessarily have been stopped, from the same instant; and his Majesty must, therefore, have dropped down, and died instantaneously (pp 266-8).

In addition to these findings, the author also noted "a transverse fissure [in the trunk of the aorta] on its inner side, about an inch and a half long, through which some blood had recently passed, under its external coat, and formed an elevated echymosis. This appearance showed the true state of an incipient aneurism of the aorta" (p. 269). No rupture of this area was described and the lungs and pleural cavities were apparently normal. Nicholls suggested that an "extraordinary and violent distension" of the aorta had occurred, possibly related to straining at a bowel movement shortly before death, and that this "must have been attended with a proportionate pressure upon the pulmonary artery, and, consequently, an increased opposition to the passage of the blood out of the right ventricle," resulting in "its [the right ventricle] bursting" (p. 270).

No additional comments were made about the coronary arteries or the walls of the ventricles. Although the explanation for the cardiac rupture is imaginative, the author gave an excellent description of hemopericardium and cardiac tamponade resulting from a ruptured ventricle, albeit involving the less frequently observed wall of the right ventricle (Fig. 1).

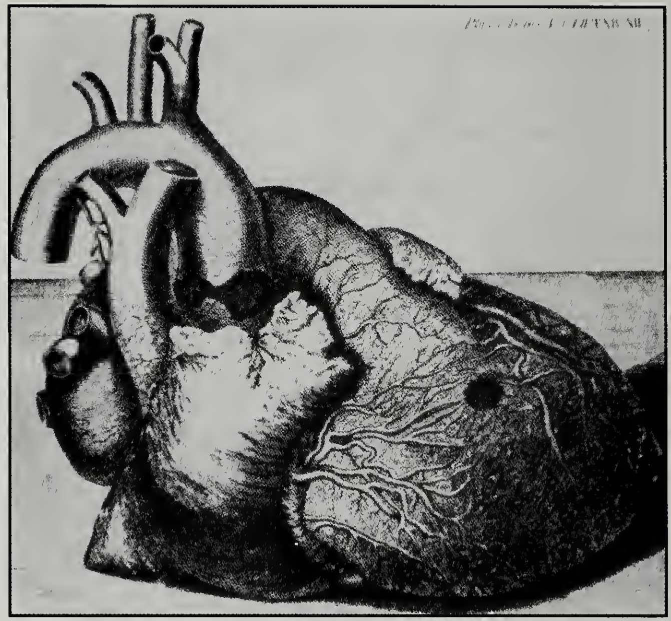


Figure 1
A print attached to the report of the autopsy findings on King George II, published in 1761, demonstrating, according to the text, "a round orifice ... in the middle of the upper side of the right ventricle of the heart, large enough to admit the extremity of the little finger" (p 268).[3]

Giovanni Battista (John Baptist) Morgagni (1682-1771), professor of anatomy at the University of Padua, and considered by many to be the founder of pathological anatomy, published his monumental word *De sedibus et causis morborum per anatomen indagatis: Libri quinque* in 1761, an English translation of which appeared eight years later.[4] In it he described several cases of cardiac rupture, some drawn from the available literature, others based upon his own observations. In one case, "a rupture of the left ventricle of the heart [was noted], near the mouth of the aorta, in a nobleman where no signs had preceded, and who being of an athletic and beautiful habit of body, had died suddenly by the side of his wife" (p. 831). No further pathological features were mentioned, although a prior comment referring to "those who died suddenly in the act of venery" (p 801)—by inference the cause in this patient—touches upon a subject that still produces much discussion when advising cardiac patients about sexual activity.

The autopsy findings in a 75-year-old woman who died after "being seiz'd with a very short stertor" were reported by Morgagni as follows:

> ... the pericardium contain'd so great a quantity of extravasted blood, partly grumous and partly fluid, that before it was cut into it resembled a spheroid, the diameter of which was nearly equal to a span.... [W]e observ'd ... [the heart] to be, in a certain part, more prominent, and almost black, from the blood which stagnated in it. This part was on the posterior surface of the heart, very near to the apex; and the fat in that part, when attentively examin'd, show'd a laceration in the middle of the blackness. The left ventricle being then open'd longitudinally on the opposite surface, we found scarce any blood therein, if you except a small coagulum adhering to that part which answer'd to the lacerated fat. This coagulum being gently remov'd led us to a round foramen of the bigness of a lentil, with which the substance of the ventricle, that was in this place naturally become very thin, had been penetrated; so that it was evident the blood had come forth

> from the heart, first, in that place, under the fat; and had afterwards insinuated itself here and there, through the membranous cells thereof; and by distending, raising up, and forcing against them, had at length easily broken through them (pp 832-3).

In addition, "bony scales" were noted lining the inner surface of the aorta, probably calcified atherosclerotic plaques. "The basilar artery was somewhat hard; as were also the other arterial branches that lie about the cerebrum" (p. 834); this, together with similar descriptions of the celiac artery and other abdominal vessels, suggests widespread arteriosclerosis. Thus the presence of the "thin" ventricle in the area of the rupture and the arteriosclerotic changes throughout the body—even without specific reference to the coronary arteries—make it likely that the patient had coronary artery disease and a myocardial infarction, possibly a ventricular aneurysm, complicated by ventricular rupture and hemopericardium.

In yet another patient who died suddenly, "the pericardium was found to be distended with concreted blood. And this had been discharg'd from the left ventricle of the heart, which a kind of little ulcer, as it were, had perforated at the apex" (pp 836-7). Although again speculative, the "ulcer," in the terminology of that period, might well have been a myocardial infarction. Morgagni also commented upon a 55-year-old man "frequently subject to rheumatic pains ... [who] was more violently seiz'd with them, on a certain day in the beginning of June, at the sternum, and in the arms" (p 838). Shortly thereafter, he died.

> ... the pericardium was found to be distended with black and coagulated blood. This blood had issu'd out of the left ventricle of the heart. through a fissure half an inch in length. and in the longitudinal direction of the ventricle, about which the fibers of the heart were seen to be destroy'd by a corrosion that was not recent (p 838).

In discussing these and other cases, and with reference to the work of Giovanni Maria Lancisi (1654-1720), a papal physician in Rome and well-known anatomist, Morgagni wrote that "among others who are liable to this kind of death, are those bodies in which ... the fibres of the heart [are] not only more languid, and not sufficiently cohering, but also deficient" (p 837). Although this description is somewhat meager and could include a variety of diseases of the heart muscle—eg, myocardial infarction, ventricular aneurysm—it does appear that it was increasingly becoming recognized that underlying myocardial disease usually had to be present for cardiac rupture to occur. However, linkage of this process with disease of the coronary arteries did not yet appear to have been made.

Morgagni anticipated later reports of the 19th and 20th centuries in noting that the preponderance of myocardial ruptures occurred in the wall of the left rather than the right ventricle:

> ... in proportion as the strength of the left ventricle is greater than that of the right, so much the more forcibly does it act upon its own parietes, especially when any obstacle opposes itself to the blood which is coming out from that cavity; so that if any part of it be, either by reason of an ulcer, or on any account whatever, whether in consequence of disease, or originally from the birth, less strong than a natural and good structure requires it to be, it cannot be equal to so great a force (p 839).

Morgagni also recorded a more unusual case of rupture of the heart caused by nonpenetrating chest wall trauma of the right ventricle that "happen'd entirely from a most violent blow that was given to the person by a carriage, which was passing him ... without any previous morbid disposition of the heart" (p 840), an event similarly reported more than 200 years later with regard to motor vehicle accidents causing blunt cardiac trauma and nonpenetrating chest wall injuries.[5,6] Apparently this patient had no underlying heart disease, as best as can be determined from the description given by Morgagni.

Cardiac Rupture as a Complication of Coronary Artery Obstruction and Myocardial Infarction

In 1799 Caleb Hillier Parry (1755-1822), a noted English physician from Bath,[7] wrote that "a principal cause of Syncope Anginosa [commonly called angina pectoris] is to be looked for in disordered coronary arteries" (p 108), calling attention to "the rigid and obstructed state of these vessels, and the disease in question" (p 109). A few years later, in 1815, Joseph Hodgson (1788-1869), a surgeon from Birmingham, England,[8] stated that "a degeneration and wasting of the muscular structure of the heart attends an extensive deposition of calcareous matter in the coronary arteries" (p 37). He described the following clinical history and autopsy findings in a 70-year-old woman:

> ... she awoke with an excruciating pain in the region of her heart.... She continued restless and extremely agitated. Her pulse fell, her countenance became pallid, and she died suddenly about six o'clock the following evening....
>
> The pericardium was distended by about twelve ounces of dark coloured coagulated blood, which had issued from a rent in the anterior part of the apex of the left ventricle. This laceration was an inch in length on the internal surface of the heart, and extended about a quarter of that distance externally. The heart abounded in soft fat, and its muscular fibers were remarkably attenuated and flaccid, more especially around the fissure in the left ventricle. The coronary arteries were incrusted with calcareous matter, and the deposition was so extensive in the one distributed to the left side of the heart, as completely to obliterate its cavity (pp 39-40).

Hodgson was also aware of the consequences of obstructed arteries in the lower extremities:

> ... experience has proved this condition of the arteries to be at least a constant attendant upon one species of gangrene, to which the extremities of old subjects are

> liable; and I have found the three principal arteries of the leg nearly obliterated by calcareous matter in two fatal cases of this disease (p 41).

With those parallel observations relating gangrene of the extremities to peripheral vascular disease and myocardial disease to coronary artery obstruction, Hodgson came very close to formulating a relationship between coronary artery obstruction, myocardial disease or infarction, and cardiac rupture.

The spectacular appearance of cardiac rupture and hemopericardium at postmortem examination prompted reports of many such cases in the medical literature during the 19th century. Most of these early accounts, a partial listing of which may be found in reviews by Schroetter[9] and von Schnebli,[10] added little to explain the pathogenesis of the problem, in spite of the suggestive clues given by Hodgson earlier.[8] However, as the century progressed, it was becoming increasingly evident from the descriptions given that cardiac rupture was usually found to be associated with pathologic changes of *both* myocardium and coronary arteries. Excerpts from representative cases will be cited to illustrate these latter observations.

In 1851 Richard Quain[11] (1816-98), author of a well-known English encyclopedia or dictionary of medicine, discussed the postmortem findings in a 68-year-old woman who had experienced chest heaviness and oppression during her life:

> … the pericardium was distended by sixteen ounces of blood partly coagulated. This blood had escaped from a rent in the anterior wall of the left ventricle one inch in length, parallel with and close to the septum. There was no very great amount of fatty growth on the heart, but in the neighbourhood of the rupture, and onwards to the apex, the muscular fibre had undergone fatty degeneration to a very marked degree (a specimen showing the oil-globules within the sarcolemma was placed under the microscope). It was of interest to observe, that other parts of the heart showed the muscular fibre to be in a remarkably healthy condition. There was thus, in fact, almost a local or circumscribed degeneration, which, it was further of interest to observe, was connected with a branch of the coronary artery distributed to this part of the heart, which was nearly occluded by calcareous degeneration of its coats….
>
> Dr. Quain said, that the present [case] was an extremely good example of the morbid condition, which had been, as he thought, very appropriately named fatty degeneration of the muscular tissue of the heart, as distinguished from the mere growth of fat on the surface, and also of one of the conditions, viz., obstructed circulation in the coronary arteries, on which this morbid state depends. It was likewise a good illustration of the connection between rupture of the heart and fatty degeneration of the fibre (pp 263-4).

In this presentation, Quain linked obstruction of a coronary artery to a pathologic change in the myocardium that he called *fatty degeneration,* an observation that would be readily identified today as an acute myocardial infarction. At the same time, he indicated a connection between the latter and cardiac rupture.

Malmsten, a clinician, and Düben, a pathologist, published one of the most detailed of these early reports associating cardiac rupture with coronary artery obstruction and a myocardial infarction. This communication, appearing originally in Swedish in 1859 and translated into English two years later,[12] described the observations at autopsy in a 66-year-old-man who died suddenly six to seven days after the initial onset of chest pain. The thoroughness of the gross anatomic examination of the heart by Düben, supplemented by microscopic description of the acute myocardial infarction, needs little amplification.

> The pericardium contained a couple of pounds of serum and about an equal quantity of coagulated black blood. Near the apex of the left side of the heart, about three-quarters of an inch from it and half an inch from the septum, was found a rupture in the cardiac wall, half an inch in length, like a chink, and filled with coagulum. It passed obliquely through the outer muscular layer of the heart and the pericardium; internally its extent could not be so accurately traced, in consequence partly of a coagulum lying close to the inside, partly of the peculiar nature of the wall. The coagulum in question lay as a round lamina, of about one and a half inches in diameter, closely adherent to the endocardium, and between the trabeculae, and in the depressions in the walls, so closely that its boundaries could not with certainty be defined, nor could it be separated from those parts. This coagulum presented on its surface turned towards the cavity of the heart the ordinary pale reddish yellow colour of fibrin, but lost it gradually out towards the wall of the heart. It was there in spots greyish yellow, light red, and dark brown; in consistence it was in one place firm, in another puriform, and passed imperceptibly into a colour and consistence exactly similar to those of the wall of the heart. The latter was, through an equal extent, about one and a half inches, softened, forming a pulpy brittle mass, the muscular filaments being, on microscopic examination, found to be destroyed and replaced by a finely granular detritus here and there mixed with fat, though they still retained their general form; in some places were found small foci, perceptible even to the naked eye, where merely a trace of connective tissue still remained and perforated the otherwise totally degenerated mass. In some parts coagula of blood derived from within were found imbedded. The necrotic change became more perceptible closer to the portion which had given way, and so coalesced with the inner part of the rupture that the extent of the latter could not accurately be determined. On examination of the coronary artery this vessel was found to be tortuous, partly in consequence of senile atrophy of the heart, partly from piecemeal dilatation, some portions of its walls being abnormally thin, and others abnormally thick. The anterior branch contained an old plug, of perfectly white colour, and completely perforated, at least in its upper extremity. It commenced some lines beyond the boundary of the softened part and extended in towards its centre, where it could no longer be traced, and where it became

> more obstructing, and was also found to be puriform.
>
> There can be no doubt that this hollowed plug was formed long ago, and gradually produced the softening I have described, as it would seem, without giving rise to any symptom. The period comprised in the history of the case given by Hr. Malmsten relates merely to the rupture; for the latter, in this instance, as in many others, occurred not in a moment, but piecemeal, and at several short intervals (pp 322-3).

Easily recognized in this report are the typical features of a ruptured left ventricle: hemopericardium, a grossly and microscopically documented myocardial infarction, and an occlusion of the infarct-related coronary artery, described as an "old plug."[12]

In 1866 the French physician and pathologist Edem-Félix-Alfred Vulpian (1826-87) described a patient with coronary artery obstruction caused by atheromatous changes and a blood clot, a myocardial infarction, and cardiac rupture at the site of the infarct, clearly suggesting an association between these findings.[13] A few years later, in a convincingly written presentation, Frederick Winsor, an 1855 graduate of Harvard Medical School, commented at a meeting of the Middlesex East District Medical Society on a 62-year-old man who, for two and one half years had been experiencing "sudden attacks of distress while walking up hill.... described as an oppression and constriction in,the carotid regions with a sense of *dread*" (p 398).[14] Following almost two weeks of more severe and frequent symptoms of angina pectoris, the patient died. At postmortem examination, in addition to a rupture in the wall of the left ventricle and a hemopericardium, the following description of the heart was given:

> The walls of the left ventricle measured nine mm. in thickness, except in the neighborhood of the rent above mentioned, where they were exceedingly thin.
>
> The color of the muscle was a reddish-brown. Under the microscope it showed a deposit of pigment in the neighborhood of the nuclei, and the dark transverse markings of the fibres were more marked than normally.
>
> In the vicinity of the rent, however, the characteristic appearance of the muscle was lost, the muscular fibres being here filled with a granular material and in many places with minute fat drops. The grayish portion of the clot above mentioned presented muscular fibres in the same degenerated condition....
>
> The walls of the left coronary artery from the point of origin to the place where the superior branch is given off were thickened, and their inner surface was covered with yellowish-white patches, some of them a roughened base, slightly sunk below the surrounding level, and others smooth and slightly raised above the level. At one point, about three cm. from the origin of the artery, one of these patches had so far protruded into the lumen of the vessel as to cause a coagulation of blood at that point, which coagulum had become adherent to the walls, thereby preventing the flow of blood through the vessel.
>
> The portion of the heart in which the rupture had occurred and in which the fibres were found degenerated corresponded to the territory supplied by the branches of this artery (p 399).

It was further indicated in this report that the severe pains occurring two weeks before death "may have resulted from occlusion of the left coronary artery; that from that time degenerative action was set up in that part of the ventricle no longer nourished by this artery; and that rupture did not occur till just before death" (p. 400). The typical gross and microscopic features of an acute myocardial infarction caused by recent thrombosis in an arteriosclerotic coronary artery, with cardiac rupture as a terminal event, are apparent in this report.

In 1884 John Lindsay Steven,[15] a clinician and pathologist from the University of Glasgow and an editor of the *Glasgow Medical Journal,* made the following comment:

> I hold, therefore, when spontaneous rupture of the heart wall is found associated with distinct disease or obstruction of a branch or branches of the coronary arteries, that the disaster has resulted from the infarction or necrosis, which has followed upon such obstruction, and not from a mere localised fatty degeneration. We know that when the circulation is cut off from any other part, necrosis is the result, and I see no difficulty in supposing that precisely the same state of matters should occur in the wall of the heart (p 423).

At this point (the latter half of the 19th century), an acute myocardial infarction secondary to coronary artery obstruction appears to have been adequately documented and established as the most common cause of spontaneous cardiac rupture. Later studies, not within the scope of the present discussion, amplified the pathology and epidemiology of this process.[16-19] In more recent years, attention has been drawn to the occurrence of hemorrhagic myocardial infarction and cardiac rupture in patients receiving thrombolytic therapy, particularly as related to the early or late administration of such treatment following the infarction.[20]

Perhaps an appropriate summary of this journey into medical history may be found in the words of James Herrick (1861-1954), an American cardiologist well known for his investigative work on coronary thrombosis, contained in his book *A Short History of Cardiology:*[21]

> The unity of coronary disease was obscured for nearly a century because the effects of such disease were often hidden under a variety of misleading captions—angina pectoris, cardiac neuroses, infarct of the myocardium, rupture of the heart, acute and chronic myocarditis, acute and chronic heart failure, fatty degeneration, partial aneurysm of the heart, pericarditis. ...
>
> It was ... [finally] realized that the Ariadne thread that guided one through the maze of angina pectoris, infarct, rupture, certain forms of pericarditis and of acute and chronic heart failure was disease of the coronary artery (pp 227-8).

Conclusion

The initial reports on cardiac rupture, beginning in the mid-17th century, focused primarily upon the dramatic findings of hemopericardium and the rent or tear of the heart wall. During the 250 years that followed, through the last half of the 19th century, careful observations and more detailed accounts gradually documented and established myocardial infarction caused by coronary artery obstruction as the principal underlying reason for this catastrophic event.

REFERENCES

1. Harvey W: A second disquisition to John Riolan, Jun., in which many objections to the circulation of blood are refuted. In: Willis R, trans. *The Works of William Harvey, M.D.* London: Sydenham Society, 1847, p 127.
2. Heberden W: *Commentaries on the History and Cure of Diseases.* London: T. Payne, 1802, pp 363-4.
3. Nicholls F: Observations concerning the body of his late Majesty, October 26, 1760. *Philos Trans R Soc Lond* 1761;52(Pt 1):265-75.
4. Morgagni JB; Alexander B, trans: *The Seats and Causes of Diseases Investigated by Anatomy.* In Five Books, Containing a Great Variety of Dissections, with Remarks. London: Millar and Cadell, 1769, Vol 1.
5. Bright EF, Beck CS: Nonpenetrating wounds of the heart—a clinical and experimental study. *Am Heart J* 1935; 10:293-321.
6. Martin TD, Flynn TC, Rowlands BJ, Ward RE, Fischer R: Blunt cardiac trauma. *J Trauma* 1984; 24:287-290.
7. Parry CH: *An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris.* London: Cadell and Davies, 1799.
8. Hodgson J: *A Treatise on the Diseases of Arteries and Veins, Containing the Pathology and Treatment of Aneurisms and Wounded Arteries.* London: Thomas Underwood, 1815.
9. Schroetter L: Spontaneous rupture of the heart. In: von Ziemssen H, ed. *Cyclopaedia of the Practice of Medicine;* Vol. 6. Diseases of the Circulatory System. New York: William Wood and Co., 1876, pp 259-67.
10. Schnebli von M. Herzrupter und Herzinfarkt in historischer und aktueller Sicht. *Schweiz Med Wochensch* 1957; 87:885-9.
11. Quain R: Fatty degeneration of the heart, causing death by rupture of the left ventricle. *Trans Pathol Soc Lond* 1851; 3:262-4.
12. Malmsten HP, Düben GWJ: Case of rupture of the heart. *Dublin Med Press* 1861; 3:322-3.
13. Vulpian E-F-A: Old cerebral softening... . Infarct of the left ventricular wall of the heart coincident with the presence of an old clot in one of the coronary arteries. Rupture of this infarct into the cavity of the ventricle and into the pericardial cavity. Haemorrhage into the pericardial cavity. In: Leibowitz JO. *The Historv of Coronary Heart Disease.* Berkeley: University of California Press, 1970, pp 184-6.
14. Winsor F: Angina pectoris with rupture of the heart—extracts from the records of the Middlesex East District Medical Society. *Boston Med Surg J* 1880; 103:398-400.
15. Steven JL: Cases of spontaneous rupture of the heart, and remarks on the pathology of the condition, etc. *Glasgow Med J* 1884; 22:413-27.
16. Krumbhaar EB, Crowell C: Spontaneous rupture of the heart: A clinicopathologic study based on 22 unpublished cases and 632 from the literature. *Am J Med Sci* 1925; 170:828-56.
17. Davenport AB: Spontaneous heart rupture—a statistical summary. *Am J Med Sci* 1928; 176:62-5.
18. Benson RL, Hunter WC, Manlove CH: Spontaneous rupture of the heart: Report of 40 cases in Portland, Oregon. *Am J Pathol* 1933; 9:295-327.
19. Levene A: Spontaneous rupture of the heart: A historical review. *Postgrad Med J* 1962; 38:334-7.
20. Honan MB, Harrell FE Jr, Reimer KA, et al: Cardiac rupture, mortality and the timing of thrombolytic therapy: A metaanalysis. *J Am Coll Cardiol* 1990; 16:359-67.
21. Herrick JB: *A Short History of Cardiology*, Springfield, Ill: Charles C. Thomas, 1942, pp 227-8.

"The Care of the Patient": A Living Testimony to Francis Weld Peabody

PETER V. TISHLER, M.D.

PEOPLE often find role models on which to pattern their social or professional development. This is certainly true for medicine, in which the apprenticeship method of training assumes almost by definition the existence and power of role models. Francis Weld Peabody has been such a figure for numerous physicians, both during his lifetime and after his death. Many of these physicians trained or worked in the medical unit established by Dr. Peabody at the Boston City Hospital, but others came under his influence through his writings only. Although Peabody was a skilled clinician and researcher, the writings that are the most pervasive are those that deal with the art of medicine. The most famous of these is a brief publication entitled "The Care of the Patient." In my essay, I trace the history of "The Care of the Patient," to demonstrate that it remains a vital and appreciated document. I also attempt to explain why this article remains influential, and why it is important in the development of the literature and practice of medicine.

Francis Weld Peabody, ca. 1925.
Photo courtesy the Francis A. Countway Library of Medicine, Boston, Massachusetts.

The Life of Francis Weld Peabody (1881-1927)

Peabody's life has been described by Oglesby Paul.[1] The facts do not adequately capture the remarkable nature of this man. He was graduated from Harvard College (1903), an average student, but he "caught fire" at Harvard Medical School (HMS). He was president of his medical school class and carried out research that formed the basis of his first five publications. He was an intern at the Massachusetts General Hospital in 1907-1908. He then took several years of postgraduate training that were distinctly different from those usually taken by a young Boston physician: nearly two years of training in medicine and pathology at the Johns Hopkins Hospital, a year studying chemistry in Germany, and fifteen months as a research and clinical resident at the new Hospital of the Rockefeller Institute in New York. In 1913, he became the first (chief) resident physician at the new Peter Bent Brigham Hospital in Boston, where he remained (with travel to China in 1914, and to Romania and France during the Great War) as a staff physician. He carried out research in a number of areas, primarily to answer pathophysiological questions generated by patients under his care. In 1921, before taking brief leave to teach in China, Peabody became the first director of the new Thorndike Memorial Laboratory and physician-in-chief of the Fourth Medical Service at the Boston City Hospital, and professor of medicine at HMS. He remained in this capacity for the rest of his life. The cause of his final illness, the early symptoms of which resulted from gastrointestinal hemorrhage, remained undetected for more than two years, despite repeated evaluation. He died of a gastric malignancy in October, 1927.

Peabody was bred to the comfortable values of the "noblesse oblige" medicine of the day. Nonetheless, he

---

PETER V. TISHLER, M.D., (AΩA, Yale University Medical School, 1962) is associate professor of medicine at the Harvard Medical School and associate chief of staff/education at the Brockton/West Roxbury Veterans Affairs Medical Center.

Copyright ©1992 by ALPHA OMEGA ALPHA HONOR MEDICAL SOCIETY, reprinted by permission from The Pharos, Volume 55, Number 3.

Send reprint requests to author at: V.A. Medical Center, 940 Belmont Street, Brockton, MA 02401.

chose to extend his world view (for example, in educational and humanitarian ventures in other countries) and explore nontraditional professional paths. He was one of a new and imaginative breed of physician who pursued a physiologic approach to the problems of medicine, a departure from the descriptive medicine of his teachers. In this role, he was a major force behind the birth of clinical investigation, in the early 1920s. Moreover, he realized the unique opportunity that a city hospital afforded to implement his notions about research. But he also remembered that his patients provided the ideas for his investigations, and that he was obliged as a physician to care for the sick. He remained ever attuned to the psychosocial aspects of being a patient, and the physician-patient relationship.

"The Care of the Patient" in Its Original Incarnation

On October 21, 1926, Professor Francis Weld Peabody delivered "to the staff and students of the [Harvard] Medical School, in the presence of many friends, a public lecture on 'The Care of the Patient'—an unpretentious and non-technical survey of the human aspects of the physician's life, which was in fact a summary of his own experience."[2] His lecture was part of a late-afternoon series of lectures with the same title for Harvard medical students, originated a year previously by a local psychologist, L. Eugene Emerson, and HMS Dean David L. Edsall, through the financial generosity of one of Dr. Emerson's patients. Although Peabody was now aware that he had an incurable cancer,[3] he nonetheless pursued the writing of this document with his customary intensity and dedication. This lecture was subsequently printed during his lifetime in the *Journal of the American Medical Association* (March 19,1927),[4] and as a hardcover book by the Harvard University Press. Hans Zinsser, Charles Wilder Professor of Bacteriology and Immunology at HMS, also combined this essay with three of Peabody's other writings in a book entitled *Doctor and Patient,* published by the Macmillan Company in 1930 and again in 1939. It gained visibility in the lay press when *Doctor and Patient* was reviewed in the *Saturday Review of Literature* by Henry R. Viets.

The written comments by Zinsser and Viets illustrate the respect with which Peabody and his writings were held by his professional contemporaries:

> [T]heir publication will serve to continue the influence of a voice that American medicine could ill afford to lose—one of clearheadedness, unsentimental idealism and the great wisdom of affectionate optimism.[5]
>
> Francis Peabody has a unique place in American medicine, for none of the younger men of recent years has made such an impression upon the medical world as the author of these few papers....
>
> ... [N]o book published in recent years is more important than this one for its value in showing ... the position of medicine today.[6]

Dr. Peabody's influence at HMS during the 1930s, as a humanist and researcher, was pervasive.[7] A second hardcover edition of "The Care of the Patient," including a photographic portrait of Peabody, was published in 1931, underwritten by the HMS class of 1907 (Peabody's class). This volume was distributed at its twenty-fifth reunion in June, 1932. In November, 1934, Joseph H. Pratt, one of Peabody's early and influential teachers at Harvard Medical School (his "scientific godfather"), made his tribute to "one we have loved and lost, Dr. Francis W. Peabody," the theme of his lecture in the annual series on the "Care of the Patient."[3] Writing to William B. Castle, in 1956, HMS graduate Frank J. Lepreau, Jr., noted, "This book *[Doctor and Patient]* was called to my attention by Dr. Henry Jackson in 1935 in his course of physical diagnosis."* Many individuals who knew Dr. Peabody, appreciated his philosophies, and had read his work now formed the nucleus of the teaching program at HMS. Nonetheless, no formal attempt was made at this time to distribute *Doctor and Patient* or "The Care of the Patient" to students.

Further Printings of "The Care of the Patient"

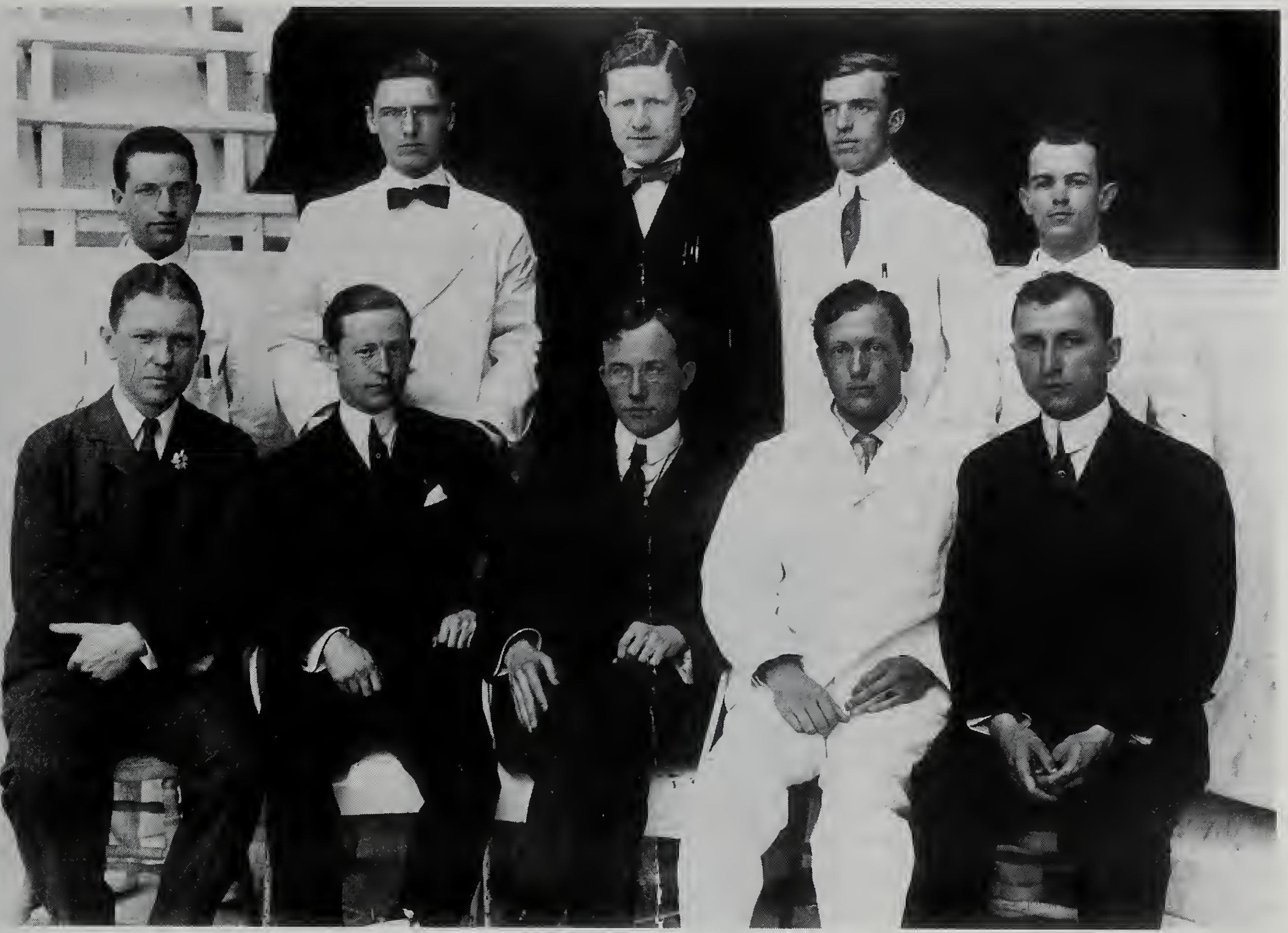
The first later reprinting of "The Care of the Patient" came not from HMS but from the Veterans Administration (VA), under the impetus of Arthur Meeker Walker (1896-1955). A 1923 graduate of HMS, Dr. Walker was probably (according to Peabody's widow, Virginia C. Shattuck) a student of Dr. Peabody. After an internship at the Massachusetts General Hospital, he joined the University of Pennsylvania Medical School and worked with Alfred Newton Richards on renal tubular physiology. After serving in the United States Army during World War II, Walker joined the VA in Washington, D.C. As chief of research and education of the Tuberculosis Service (1950-55), and editor of the VA "Technical Bulletin" Series 10, Dr. Walker reprinted "The Care of the Patient" on December 20, 1952 (Technical Bulletin 10-85). Printed by the Government Printing Office, it was distributed nationally, to VA hospitals and to other individuals. Dr. Walker told Mrs. Shattuck that the distribution reached about 14,000 physicians.

Dr. Walker's editorial comment accompanying this reprinting is noteworthy:

> It is almost precisely twenty-five years since, at the age of 46, Francis Peabody died. In his own

---

*Lepreau to Castle, January 20,1956, Fall River, Massachusetts.



The medical staff of the Johns Hopkins Hospital, 1908-1909. Francis W. Peabody is on the far left of the front row.
This and following photo courtesy of the author.

> person, he exemplified what he steadfastly advocated: the importance of art as well as science in the practice of medicine. He continued to teach while he was clearly confronted by death. He continued to write when he was no longer able to teach. This article is one of his last writings. It is reproduced here because it expresses the understanding and compassion of a true physician, an expression which is capable, if accepted, of inspiring a generation which could not know him as did those who came directly under his influence. But let his words speak for him; they are the words of a man long dead, but words which still live and of which medicine is still in need.[8]

Impetus for reprinting Dr. Peabody's work came from another source in December, 1955. Thomas Lowry, a house officer on the Harvard Medical Services at the Boston City Hospital from 1935 to 1937 wrote to Dr. Castle to ask for advice and assistance in reprinting *Doctor and Patient.* He stated:

> It seems to me that the wisdom and philosophy contained in this book should, if possible, be preserved and transmitted to as many of the present generation of doctors as possible. I believe that it would be an ideal present to give to each member of any graduating class of medical students, or to interns upon the completion of their internship, or to residents in medicine at the completion of their training period.*

Dr. Castle, at this time director of the Harvard Medical Unit at the Boston City Hospital, discussed the issue with HMS Dean George Packer Berry. Dean Berry was pessimistic about stimulating interest in or finding funds for the project. Fortunately, Dr. Lowry's letter was followed in January 1956 by the aforementioned letter to Dr. Castle from Dr. Lepreau, and one from Morton Hamburger, of the University of Cincinnati, that raised similar questions. Dr. Castle asked his Thorndike colleague Edward H. Kass to sample faculty and student opinion concerning reprinting *Doctor and Patient.* Dr. Kass wrote to Dr. Castle, "I have asked the 40-50 members who appeared at the last meeting of the Boylston Society [the student oratorial society of which he was president] for their reactions. Only about six [student members] had heard of the book (I blush to confess that some of those who were unfamiliar with it were former students of mine) and only three had read it."* Dr. Kass suggested that HMS reprint only "The Care of the Patient." Dr. Castle asked Mrs. Shattuck for her support for (and possible underwriting of) reprinting "The Care of the Patient." Her second husband, HMS Professor Emeritus George Cheever Shattuck, expressed the family's enthusiasm for the effort and contributed a brief foreword for the publication. Publicity for the printing was generated by both Dr. Castle, who wrote some thirty-eight "friends among the Professors of Medicine," and Dean Berry, at a meeting of the Association of American Medical Colleges. The printing of 20,000 copies by the Harvard University Press was completed in the summer of 1956. Copies were distributed to physicians at such institutions as Iowa (William B. Bean), Yale (Paul B. Beeson), Johns Hopkins (A. McGehee Harvey), Washington University (Carl V. Moore), Alabama (Tinsley R. Harrison), Duke (Eugene A. Stead, Jr.), Washington (Robert H. Williams), and many others. By October, 1956, the supply was nearly exhausted.

---

*Lowry to Castle, December 1,1955, Minneapolis, Minnesota.

Stimulated directly by the VA publication of Dr. Peabody's work, the Department of Medicine at the University of Maryland had begun to prepare its own copy of "The Care of the Patient." Theodore Woodward, chairman of the department, gave copies to each member of the second-year class from 1956 until his retirement in 1981. Thereafter, his son-in-law, Mark Applefeld, has continued the same tradition in his role as director of the course on physical diagnosis.

At HMS copies were given to all entering medical students for a number of years. Copies were also distributed to interns and residents at the affiliated Harvard teaching hospitals, including the Departments of Medicine at the Massachusetts General, Beth Israel, and Boston City Hospitals, and possibly other programs:

Drs. Lepreau and HMS faculty member Perry J. Culver convinced Macmillan to reprint *Doctor and Patient* in 1973 or 1974. These two Harvard alumni also underwrote the publication of a book entitled *The Art of Medical Care and Caring* in about 1982. This volume, containing the entire contents of *Doctor and Patient* plus articles by HMS distinguished alumni Frederick Cheever Shattuck and J. Engelbert Dunphy, was given to Harvard medical students at graduation for a time.

In the mid-1970s, the Department of Medicine at the Mount Auburn Hospital in Cambridge, Massachusetts, printed copies of "The Care of the Patient," and has since distributed them to house staff and Harvard medical students during their medical clerkships. "The Care of the Patient" was also reprinted in the *Journal of the American Medical Association* in 1984, as a Landmark Article.[9] In 1986, the Medical Service of the Brockton/West Roxbury Veterans Administration Medical Center reprinted Shattuck's 1956 edition. Copies are distributed to Harvard medical students on the wards as clinical clerks, and to medical house officers on rotation from the training programs at the Beth Israel and the Brigham and Women's Hospitals.

Has "The Care of The Patient" Been Relevant?

A few examples will suffice to indicate that "The Care of the Patient" is still read, and its message is still relevant. HMS alumnus Henry H. Work wrote that he still has his copy, bought while he was a student, and that "it is a constant reference."[10] In a letter to me, he noted that he always encourages his first-year students at the Georgetown University School of Medicine "to read Peabody and we discuss it."* David and Pauline Rabin wrote a commentary accompanying the reprinting of "The Care of the Patient" in 1984.[11] Dr. Pauline Rabin said that Dr. David Rabin, who was gravely ill at the time, found Peabody's writing "inspirational." The work was also reprinted by John D. Stoeckle, professor of medicine at HMS, in a discussion of good physician-patient relations in the anthology *Encounters Between Patients and Doctors.*[12] "The Care of the Patient" was a focus of a recent discussion of the interrelationships of medicine and religion.[13] Lloyd H. Smith, Jr., quoted Peabody in discussing the interaction of students and patients in his introductory essay in *Cecil Textbook of Medicine.*[14] Robert E. Kravetz has written about his own illness, and noted that he is now much more aware of the "art of medicine." "The new direction that I have chosen in medicine is to learn more about 'becoming a better practitioner of, and teacher of humanism.' The ... words of Francis W. Peabody ... have taken on a very personal meaning for me."[15]

Why has this document been so frequently reprinted and quoted, and otherwise remained so visible? Perhaps the most important reason relates to the character of Francis Weld Peabody himself. He was soft-spoken; unassuming; humble; compassionate; and intensely dedicated to the care of patients, to the training and nurturing of other professionals, and to the development of clinical investigation. Apparently, he was the charismatic embodiment of the virtues about which he wrote.[16] Thus, many of his colleagues and students *wanted* to perpetuate his memory (Zinsser, Walker, his medical school classmates, et cetera). Further impetus came from the early members of Peabody's Thorndike Memorial Laboratory and the Harvard Medical Unit at the Boston City Hospital. The efforts of this generation—which included some well-known individuals, with their own charisma—publicized "The Care of the Patient" for the recent generations. For many of Peabody's descendants at the Boston City Hospital, his name still has mystical, parent-like connotations.

---

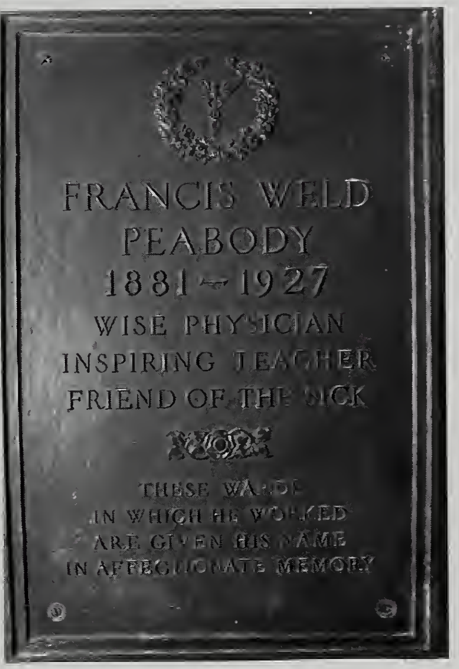
*Kass to Castle, January, 1956, Boston, Massachusetts.

*Henry H. Work 1991: personal communication.

The work strikes a particularly respondent chord in teachers. They are familiar with Peabody's goals as an educator. He noted that the "proper employment of the fundamental sciences in the study of disease has become an essential for good medical practice."[17] The educator's role, however, was to teach that "over and above this, there must be something which correlates the scientific findings with the actual problems of the individual case—the art of medicine, which carries us beyond the patient to the man."[17] Peabody took his role as a teacher and mentor seriously:

> I feel that probably I accomplish most in the long run ... by accepting as my major function that of helping my students and assistants. Much greater contribution to the advancement of medicine will come from the training of these men than through my own individual efforts.[18]

It is not surprising that the first memorial that was established at HMS by his colleagues (in 1928) was a fellowship. As stated in the Dedication, the fellowship is a vehicle with which properly trained young men could be enabled to follow in his footsteps and fit themselves in the manner he thought best for the practice and teaching of clinical medicine.



This tablet hung in the Burnham Building ("Peabody 1"), Boston City Hospital, from 1935 to 1990.

Physician-educators who are committed to the teaching of medicine agree with Peabody's contention in "The Care of the Patient" that "medicine is not a trade to be learned but a profession to be entered," and that this sense of dedication must be communicated. They identify with Peabody's commitment. Dr. Work suggested that Peabody articulated a "message of true concern for and, hopefully, empathy with patients," virtues he fears are lost nowadays in medical school curricula.* Peabody's caring yet scientific approach to patients is a particularly relevant contemporary model, because of the sophisticated technologies that often constitute current therapy. He demonstrated that both attitudes are possible. Indeed, "The Care of the Patient" has many lessons for the physician-student, at any stage of education or professional life. Caring physicians and teachers transmit his values as their own.

"The Care of the Patient" as Autobiography

Personal thoughts appear to be accompanied by personal experiences in "The Care of the Patient." Peabody described a patient "who entered the hospital with a history of nausea and discomfort in the upper part of the abdomen after eating."

> The history suggests a possible gastric ulcer or gallstones, and ... she is given a test meal, gastric analysis and duodenal intubation, and roentgen-ray examinations are made of the gastro-intestinal tract and gall-bladder. All of these diagnostic methods give negative results; that is, they do not show evidence of any structural change."[4,p.878]

Is this coincidence, or was it related to his own recent experiences with the subtle early manifestations of his own stomach cancer?

Literature addressing the experience of illness was decidedly unusual during Peabody's lifetime. William B. Ober has noted that literary writings by patients (including physicians) about their illnesses were not much in evidence until well into the twentieth century.[19] In 1924, Alfred and Martin Grotjahn began a collection that ultimately included 137 brief autobiographical reports of disease (some fatal) in physicians.[20] With rare exception, these reports were descriptive, matter-of-fact, and in no way introspective. Thus, Peabody's account may have stood alone at the time. Against the background of the formality of the recent Victorian era in general and medicine in particular, Peabody showed immense courage and, once again, innovation in revealing his soul in print. In fact, Peabody had a sustained interest in the emotional and psychological aspects of human nature. He had participated in the convivial gatherings of a group called the Olympians at the home of Morton Prince, an energetic, persuasive physician who introduced the field of abnormal psychology in Boston. Comprised of psychiatrists, biologists, psychologists, and philosophers, the Olympians met to discuss "questions of general scientific import."[21,22] Peabody was drawn to these meetings through his interest in such questions, which undoubtedly included neuropsychologic issues. His writings on the psychosomatic aspects of illness, so prominent in "The Care of the Patient," may have evolved from this exposure.

One suspects that Francis Weld Peabody's personal experiences with illness and hospitalization resembled in some part that

---

*Henry H. Work 1991: personal communication.

> poor fellow who has just been jolted to the hospital.... A string of questions about himself and his family has been fired at him, his valuables and even his clothes have been taken away from him, and he is ... miserable, scared, defenseless and, in his nakedness, unable to run away.... Here, is a worried, lonely, suffering man.[4,pp 880-81]

Peabody almost certainly selected his examples and crafted his text intentionally, as his means of personalizing his message. Stoeckle has suggested that Peabody "may well have been aware of how the dying feel rejected."[23] He may thus have written one of the first autobiographical analyses by a physician of his emotional response to his own fatal ilness. It was a plea from an insightful physician to future physicians. His legacy is a complete, universal definition of the art of caring:

> The good physician knows his patients through and through, and his knowledge is bought dearly. Time, sympathy and understanding must be lavishly dispensed, but the reward is to be found in that personal bond which forms the greatest satisfaction of the practice of medicine. One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient.[4, p 882]

REFERENCES

1. Paul O: The Caring Physician: The Life of Dr. Francis W. Peabody. Boston, Massachusetts, Francis A. Countway Library of Medicine, 1991.
2. Peabody FG: Francis Weld Peabody, 1881-1927, A Memoir. Cambridge, Massachusetts, Riverside Press, 1933, p 64.
3. Pratt JH: The personality of the physician. *N Engl J Med* 1936; 214:364-70.
4. Peabody FW: The care of the patient. *JAMA* 1927; 88:877-82.
5. Zinsser H: Introduction. *In:* Peabody, FW: Doctor and Patient, pp. ix-xiii, New York, Macmillan Co., 1930, p xii.
6. Viets HR: Book review: Doctor and Patient. *Saturday Review of Literature* 1930; 7:422.
7. Roberg N: Internal medicine in the 1930s. *JAMA* 1988; 260: 3645-6.
8. Walker AM: Editor's Note: Reprinting of Peabody, FW: The care of the patient (1927). Veterans Administration Technical Bulletin, TB10-85, Washington, D.C., Government Printing Office, December 20, 1952.
9. Peabody FW: The care of the patient (1927). Reprinting *JAMA* 1984; 252:813-8.
10. Work HH: Psychiatrists left out? Letter. *Harvard Medical Alumni Bulletin,* Winter 1990-1991; 64(3):3.
11. Rabin PL and Rabin D: The care of the patient: Francis Peabody revisited. *JAMA* 1984; 252:819-20.
12. Peabody FW: The care of the patient (1927). Reprinted in: Stoeckle, JD, ed: Encounters Between Patients and Doctors, pp 387-401. Cambridge, Massachusetts, MIT Press, 1987.
13. Gomes PJ, Austin D, Sinha A, et al: An agreeable conversation on the border of religion. *Harvard Medical Alumni Bulletin* , Winter 1990; 63(3):12-21.
14. Smith LH, Jr: Medicine as an art. *In:* Wyngaarden JB, and Smith LH, Jr, eds: Cecil Textbook of Medicine, 18th ed., pp 1-5. Philadelphia, W. B. Saunders., Co., 1988.
15. Kravetz RE: Bleeding ulcer. *In:* Mandell H, and Spiro H, eds.: When Doctors Get Sick, pp. 429-37. New York, Plenum Medical Book Co., 1987, p 436.
16. Zinsser H: Obituary: Francis W. Peabody. *Boston Med Surg J* 1927; 197:1288-92.
17. Peabody FW: The patient and the man. Presentation to New York Academy of Medicine, November 22,1922, unpublished. Reprinted in Paul, O: The Caring Physician: The Life of Dr. Francis W. Peabody, pp 148-54. Boston, Massachusetts, Francis A. Countway Library of Medicine, 1991, p 154.
18. Peabody FW: The soul of the clinic. *JAMA* 1928; 90:1193-97, pp 1196-7.
19. Ober WB: Foreword. *In:* Mandell H and Spiro H, eds.: When Doctors Get Sick, pp vii-ix. New York, Plenum Medical Book Co., 1987.
20. Grotjahn A: Arzte als Patienten. Leipzig, Georg Thieme, 1929.
21. Murray HA: Morton Prince: Sketch of his life and work. *J Abn Soc Psychol* 1956; 52:291-95, p 294.
22. Roback AA and Kieman T: Pictorial History of Psychology and Psychiatry. New York, Philosophical Library, 1969, pp 237-39.
23. Stoeckle JD; Introductory comments. In: Stoeckle JD, ed: Encounters Between Patients and Doctors, Cambridge, Massachusetts, MIT Press, 1987, p 383.

Acknowledgments: This work is dedicated to the memory of William B. Castle, late director of the Harvard Medical Unit at the Boston City Hospital. Dr. Castle, in continuing the tradition of Francis Weld Peabody, was an inspiration to all his students.

The author thanks the following physicians for information (personal letters or conversations): Frank J. Lepreau, Jr., James E. C. Walker, Theodore Woodward, Mark Applefeld, Henry H. Work, Pauline Rabin, and Robert E. Kravetz. He is also indebted to Oglesby Paul for detailed information about Dr. Peabody, and for criticism of this manuscript; Elin L. and Richard J. Wolfe of the Countway Library, Harvard Medical School, for providing discussion and unpublished source material from the files of Dr. Francis W. Peabody, the Twenty-fifth Reunion of HMS Class of 1907, and Dr. William B. Castle; Amalie M. Kass for criticism and the reality testing of a seasoned historian; Gerard Shorb, research associate at the Alan Mason Chesney Medical Archives, the Johns Hopkins Medical Institutions for identifying the individuals in the photograph of Dr. Peabody and his Hopkins co-workers; and Sigrid L. Tishler for ideas and editorial criticism.

Deaths and Hospitalizations from Chronic Liver Disease and Cirrhosis—United States, 1980-1989

CENTERS FOR DISEASE CONTROL, ATLANTA

IN 1989, chronic liver disease,* including cirrhosis, was the ninth most frequent cause of death in the United States.[1] Periodic analysis of trends and factors related to preventable death and hospitalization for chronic liver disease may be used to target prevention and control programs. This report examines national trends in death and hospitalization rates and state-specific death rates for chronic liver disease using data from CDC's National Center for Health Statistics' multiple-cause-of-death file and the National Hospital Discharge Survey (NHDS).

From 1980 through 1989, the age-adjusted death rate† for chronic liver disease decreased 23%, from 13.5 to 10.4 per 100,000 persons (Fig. 1). During this period, rates for men were more than two times higher than for women, and rates for blacks were more than 50% higher than for whites.§ Death rates for each of these groups declined steadily during this period.

In 1989, chronic liver disease was the underlying cause of death for 26,720 persons (Table 1) and a contributing cause of death for an additional 14,101 persons. Among deaths for which chronic liver disease was the underlying cause, 46.1% were diagnostically associated with alcohol (ie, alcoholic fatty liver, acute alcoholic hepatitis, alcoholic cirrhosis of the liver, and alcoholic liver damage—unspecified); 2.9%, with chronic hepatitis; 1.5%, with biliary cirrhosis; and 49.5%, with unspecified conditions and no mention of alcohol (ie, cirrhosis of the liver without mention of alcohol, other chronic nonalcoholic liver disease, and unspecified chronic liver disease without mention of alcohol).

Age-specific death rates increased with age for men in the 35-44-year through 65-74-year age groups (from 15.2 to 49.0 per 100,000 men) and for women in the 35-44-year through 75-84-year age groups (from 4.8 to 26.7 per 100,000 women) (Table 1). State-specific age-adjusted death rates of chronic liver disease in 1989 varied more than fivefold, from 6.1 per 100,000 population (for Idaho) to 31.5 per 100,000 (for the District of Columbia). The median rate was 9.6 per 100,000.

Chronic liver disease was also an important, although diminishing, cause of hospitalizations during 1980-1989. The age-adjusted hospitalization rate of chronic liver disease decreased 44% during this period (from 50.6 to 28.2 per 100,000) (Fig. 1). Rates for women were generally one third lower than for men, and for both, declined steadily throughout the decade. For most years, rates for whites were 20%-30% lower than rates for blacks.

Chronic liver disease appeared as the first-listed diagnosis in an estimated 72,232 hospitalizations in 1989 (Table 2). Among these hospitalizations, 49.3% were diagnostically associated with alcohol, 10.5% with chronic hepatitis, 1.8% with biliary cirrhosis, and 38.3% with unspecified conditions and no mention of alcohol. Chronic liver disease was also listed as a diagnosis (other than first-listed) in an additional 218,156 hospitalizations.

Age-adjusted hospitalization rates of chronic liver disease in 1989 were 38% higher for men than for women (33.1 versus 23.9 per 100,000) and 27% higher for blacks than for whites (30.1 versus 23.7 per 100,000). Rates were successively higher in each age group from 35-44 years through 55-64 years for both men and women (from 40.9 to 96.5 per 100,000 and from 30.1 to 88.9 per 100,000, respectively) and decreased sharply after this age.

Reported by: Chronic Disease Surveillance Bureau, Office of Surveillance and Analysis, National Center for Chronic Disease Prevention and Health Promotion, CDC.

Editorial Note: Most specific types of chronic liver disease in the United States are preventable.[2] The findings in this report indicate a steady decline in rates of hospital-

---

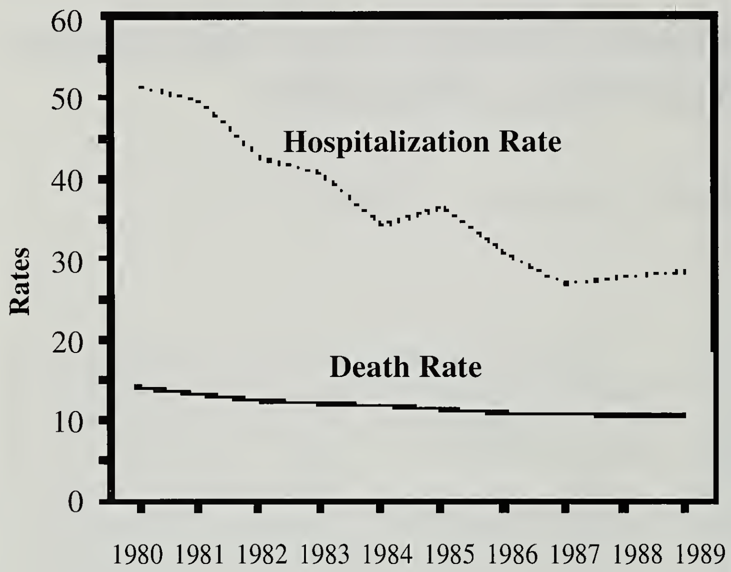
* International Classification of Diseases, Ninth Revision, code 571.

† Based on the underlying cause of death. Intercensal population estimates were used to calculate age-adjusted rates standardized to the 1980 U.S. population.

§ Estimates are presented by race to address the national health objectives for the year 2000 to reduce cirrhosis deaths in special populations. Estimates are not presented for races other than black and white because numbers were too small for analysis.

Morbidity and Mortality Weekly Report, Vol.41, Nos. 52 & 53, January 8, 1993.

Figure 1.
Hospitalization and death rates* of chronic liver disease—United States, 1980-1989.



ization and death from chronic liver disease during the 1980s. The variation in state-specific age-adjusted death rates suggests underlying regional differences in the occurrence of chronic liver disease and related risk factors. These findings may be used to target prevention and treatment programs and in the design of further epidemiologic research.

The findings in this report are subject to at least two limitations. First, because NHDS data do not distinguish initial from recurrent hospitalizations for a given person, these results represent the number of hospitalizations rather than the number of persons hospitalized for chronic liver disease. Thus, the declines might reflect a decline in the number of persons with chronic liver disease or in fewer hospitalizations among those with chronic liver disease, or some combination of both. Second, for both hospitalization and death certificate data, alcohol-related diagnoses may be underreported.

Despite these potential limitations, the declining hospitalization and death rates reported here may indicate a true decrease in the underlying occurrence of chronic liver disease as a result of decreases in the prevalences of major risk factors (eg, heavy alcohol use). In the United States, heavy alcohol use is considered the most important risk factor for chronic liver disease; even among deaths coded as chronic liver disease with unspecified conditions and no mention of alcohol, approximately 50% are thought to be due to alcohol use.[3] Thus, decreasing hospitalization and death rates may reflect, in part, the decline in per capita alcohol consumption from 1977 through 1989.[4] These findings also are consistent with data from CDC's Behavioral Risk Factor Surveillance System that have shown a greater proportion of heavy drinkers among men than women and that alcohol consumption is inversely related to age.[5] Strategies for reducing per capita consumption of alcohol include price controls (eg, increased taxes on alcohol), control of the physical availability of alcohol, changes in legal accessibility, information and education programs, health warn-

Table 1.

Age- and sex-specific death rates* of chronic liver disease†—United States, 1989

	Men		*Woman*		*Total*	
Age- group (yrs)	*No.*	*Rate*	*No.*	*Rate*	*No.*	*Rate*
35-44	2,720	15.2	888	4.8	3,608	9.9
45-54	3,389	28.1	1,345	10.6	4,734	19.1
55-64	4,521	44.6	2,238	19.8	6,759	31.5
65-74	3,941	49.0	2,562	25.5	6,503	36.0
75-84	1,732	47.2	1,611	26.7	3,343	34.5
⩾85	302	35.7	402	18.4	704	23.3
All-ages	**17,325**	**14.4**	**9,395**	**7.4**	**26,720**	**10.8**
Crude rate		*14.4*		*7.4*		*10.8*
Adjusted rate[§]		*14.7*		*6.6*		*10.4*

*Per 100,000 persons.
†*International Classification of Diseases, Ninth Revision,* code 571.
[§]Age-adjusted to the 1980 U.S.standard population.

Table 2.

Age- and sex-specific hospitalization rates* of chronic liver disease†—United States, 1989

	Man		*Women*		*Total*	
Age group (yrs)	*No.*	*Rate*	*No.*	*Rate*	*No.*	*Rate*
35-44	7,325	40.9	5,523	30.1	12,848	35.4
45-54	8,877	73.7	6,947	54.6	15,824	63.9
55-64	9,789	96.5	10,065	88.9	19,854	92.5
65-74	6,146	76.4	6,653	66.3	12,799	70.8
75-84	3,061	83.4	1,618	26.8	4,679	48.2
≥85	586	69.4	881	40.4	1,467	48.5
All ages	**39,717**	**33.0**	**32,515**	**25.7**	**72,232**	**29.3**
Crude rate	*33.0*		*25.7*		*29.3*	
Adjusted rate§	*33.1*		*23.9*		*28.2*	

*Per 100,000 persons.

†International Classification of Diseases, Ninth Revision, code 571.

§Age-adjusted to the 1980 U.S. standard population.

ing labels, targeted health-promotion programs, and related activities.[6]

Hepatitis B and C viruses are also important risk factors for chronic liver disease,[7] and their relative contribution to chronic liver disease, alone and in combination with alcohol, requires further study. A comprehensive vaccination strategy for eliminating hepatitis B virus transmission and its sequelae in the United States has been recommended.[8] Other potential risk factors include certain drugs, industrial chemicals, and less common infectious agents.

An estimated 90% of deaths attributed to cirrhosis is preventable.[2] The national health objectives for the year 2000 include reducing cirrhosis deaths to no more than six per 100,000.§[9] The findings in this report underscore that efforts to decrease mortality associated with chronic liver disease will have to be intensified if this objective is to be met.

---

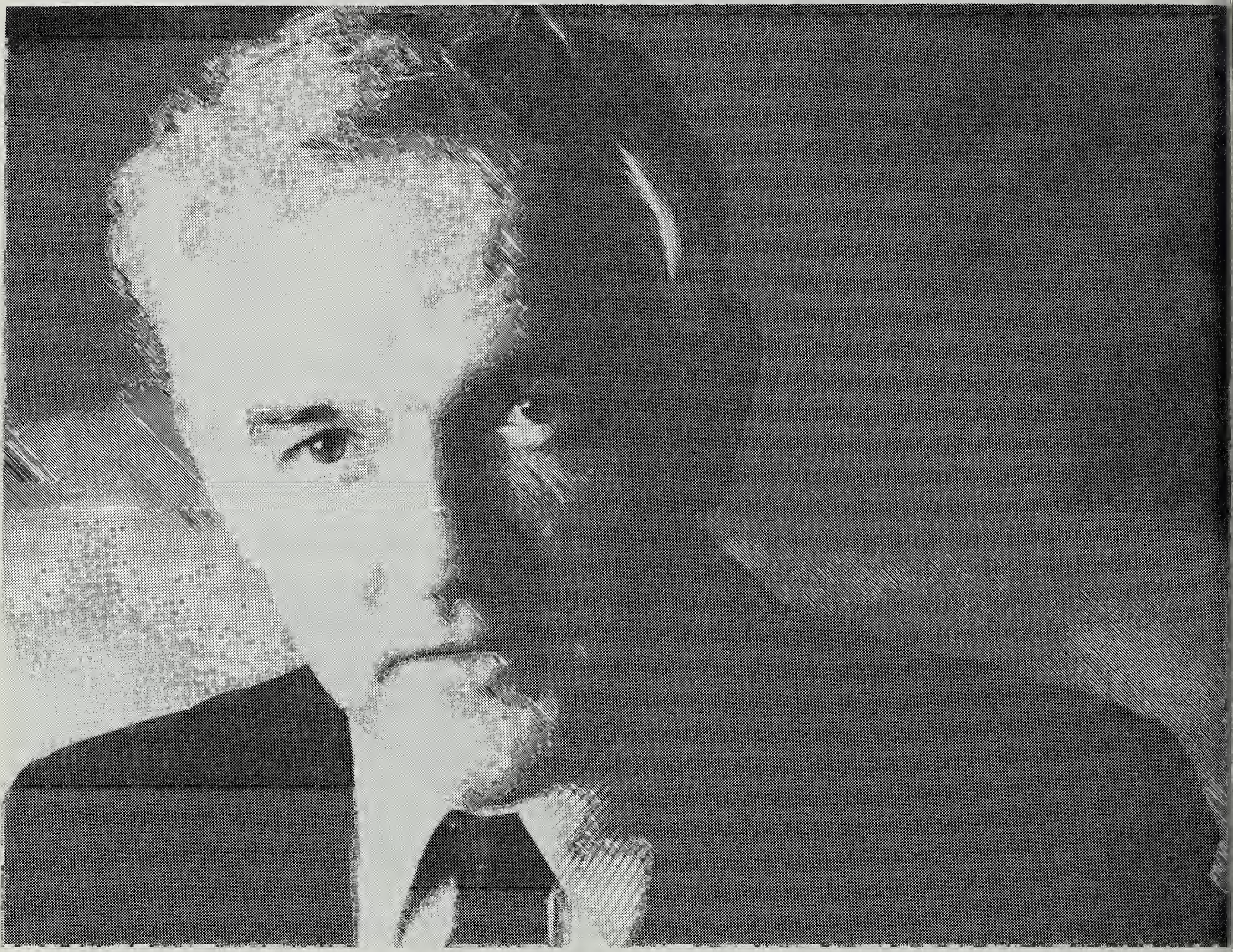
§Age-adjusted to the 1940 U.S. standard population.

REFERENCES

1. NCHS. Advanced report of final mortality statistics, 1989. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1992. (Monthly vital statistics report: Vol. 40, No. 8, Suppl 2).
2. Rothenberg RB, Koplan JP: Chronic disease in the 1990s. *Ann Rev Public Health* 1990:11:267-96.
3. Shultz JM, Parker DL, Rice DP: ARDI: alcohol-related disease impact software [Software documentation]. Atlanta: US Department of Health and Human Services, Public Health Service, CDC, 1989.
4. National Institute on Alcohol Abuse and Alcoholism. Apparent per capita consumption: national, state, and regional trends 1977-1989. Rockville Maryland: US Department of Health and Human Services, Public Health Service, 1991; DHHS publication no. (ADM) 281-89-0001.
5. Anda RF, Waller MN, Wooten KG, et al. Behavioral risk factor surveillance, 1988. In: CDC surveillance summaries (June). *MMWR* 1990-39 (No. SS-2): 1-21.
6. Rankin JG, Ashley MJ: Alcohol-related health problems. In: Last JM, Wallace RB, eds. Maxcy-Rosenau-Last public health and preventive medicine. 13th ed. East Norwalk, Connecticut: Appleton and Lange, 1992.
7. Hurwitz ES, Neal JJ, Holman RC, et al: Chronic liver disease deaths associated with viral hepatitis in the United States [Abstract]. In: Program and abstracts of the Seventh National Conference on Chronic Disease Prevention and Control. Atlanta: US Department of Health and Human Services, Public Health Service, CDC, 1992:85.
8. ACIP. Hepatitis B virus: A comprehensive strategy for eliminating transmission in the United States through universal childhood vaccination—recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR* 1991.40) No. RR-13).
9. Public Health Service. Healthy people 2000: National health promotion and disease prevention objectives—full report, with commentary. Washington, DC: US Department of Health and Human Services, Public Health Service, 1991: DHHS publication no. (PHS) 91-50212.

"Filling out insurance forms gives my patients a pain in the neck."

—Dr. Patrick J. Carolan, Orthopedics, Bridgeport, CT



"Good medical care was once as easy as seeing your doctor. Complications set in. Forms to fill out. Deductibles. Hidden costs. Partial coverage. Non-medical intrusions into the patient-physician relationship.

"Doctors decided to do something about it. Through our State Medical Society, we first created **CSMS-IPA,** Connecticut's only *statewide* IPA. Then we launched **M.D. Health Plan,** based on a simple principle: The best medicine for *patients* is practiced by *physicians*— not insurance companies, not benefit managers, not cost analysts.

"It worked! Now the fastest growing health plan in Connecticut and the choice of over 68,000 patients, our company is 100% owned and 100% directed by practicing physicians. Why not?"

Patrick Carolan, M.D., President, CSMS-IPA

If *you* wish to join the 3,000 CSMS physicians who now participate, call us at one of the numbers below.

M.D. HEALTH PLAN

6 DEVINE STREET • NORTH HAVEN, CT 06473 • (203) 776-5759 • 1-800-345-9272

Editorial

An Outgoing, Delegate's Point of View

SEVERAL years ago I wrote an article for *Connecticut Medicine* entitled, "A Newcomers View of the AMA Process." Having just completed 12 years at this post, I thought it would be worthwhile to look at some of the changes that have taken place in the interim to see if my viewpoints have varied greatly during this period of time with the AMA. At first I stood in awe of not only the great responsibilities of the AMA House of Delegates, but also of the great efficiency and democracy with which it was run. I am happy to say that these attributes are even more true today than at that time. When you take your seat at these auspicious occasions, you hope to reflect credit upon Connecticut. You are surrounded by 400 plus delegates from other states and specialty societies and thus can actually feel intimidated at first.

What other aspects of the AMA were most impressive? It is heartening to know that by our election process, various committees and councils of the AMA are served by members who are all tremendously qualified for their jobs. At the time of the elections and appointments to these positions, it is hard to cast a vote in view of the great qualifications and experiences of the candidates. By no means are these simply "figure head" type positions. The members of these councils deal with the most delicate and also the most difficult matters that face us as practicing physicians. I have been greatly impressed by the way in which some of the most difficult problems that confront medicine are handled by referral to these councils and to the Board of Trustees. They, in turn, have issued well thought out comprehensive reports in matters referred to them.

In the years that I have served on this delegation, the onslaught against the private practioner has been unfair and has progressively worsened. Apparently the saying still holds true, that the patients revere their own doctors but have a great dislike for "medicine." It is certainly a paradox, that at a time when world powers elsewhere have abandoned the socialist type schemes that have not served them well that we should be heading in this wrong direction.

In 1965 we were assured that the Medicare program would not inflict major government regulations upon us that would affect the way we practice medicine. This obviously proved untrue. The RBRVS Program (Resource Based Relative Value Study) was an attempt by the government to improve access to care and at the same time allow a pluralistic system of medicine to survive. In an effort to "out do Santa-Claus" the opposing candidate, it would seem that medicine has been placed on the auction block. This has lead to a plethora of government intrusion policies into the practice of medicine. The AMA has definitely blunted some of these intrusions, and is working hard to have some of the other programs make sense. To mention a few such concerns, at the recent interim meeting of the AMA the matter of unfair, unrealistic, and costly programs such as CLIA (Clinical Laboratory Improvement Act) and OSHA (Occupational Safety Health Administration) have been addressed. The "self-referral" situation has been handled in an admirable way. The incoming Clinton administration seems headed in the direction of some type of "managed-care" approach hopefully to improve medical care access. The AMA has accepted this, again under the heading of "the pluralistic approach." The caveat mentioned that, to date, managed-care systems have not proven their worth consistently. The serious AIDS situation continues to be dealt with comprehensively by the AMA. This journal has itself excerpts from these excellent AMA reports.

We know full well "medicine" cannot rest on the laurels of past accomplishments (prolongation of life, technical improvements in delivery of care, and many treatments of serious illness that were unavailable only a few years ago). Therefore government intrusion, unfairness by the media, and some significant disagreements with former allies (American Hospital Association and the pharmaceutical industry) have all required careful consideration by the House of Medicine.

Although a high degree of certification of physicians as to general competency has already been occurring by virtue of hospital accreditation, acceptance into medical associations, HMOs, specialty society certification, etc., this apparently has not been enough for the general public. A national clearing house has already been in existence to help identify the "bad doctor." In answer to this hue and cry for closer scrutiny of the profession, the AMA worked with others to develop the National Practicioners Data Bank (NPDB). The AMA is now working to make this more meaningful since at this time many aspects of the NPDB have been subverted. The AMA has been recognized by many as being proactive and it has certainly been timely in acknowledging and responding to health matters as well as to challenges to the profession. To the many doctors who feel that the AMA "hasn't done much for me lately," I can only say that they are not aware of the involvement at all levels by the AMA in interacting with government agencies and medical organizations. During my term of office, I believe the AMA has been successful in casting off the image of "an old boy's club" and being out of step with the times. During the past years the students' and residents' sections have earned great respect from the House of Medicine.

The resolutions coming from these sections, as well as the Young Physician Section, has kept the AMA not only in step with the times but also anticipatory in the concerns of medicine and regulatory matters of health care concern.

In order truly to be the "umbrella" under which the AMA attempts to deal with all points of view regarding medical matters, the Hospital Medical Staff Section was formed several years ago. This has lead to a profusion of excellent resolutions which have been debated and highly considered by the House of Delegates. Apparently it is a little known fact that a large majority of reports and resolutions dealt with by the delegates are of a "probono" nature. Other resolutions often reflect policies that allow the physician the freedom to practice. This freedom has characterized our profession and its envied world medical leadership.

The biennial meeting of the AMA can never be considered perfunctory or dull. There is a great sense of excitement and feeling of need to promote and comment on matters proposed by the previously mentioned excellent reports of the various councils, as well as the resolutions from other states and organizations. More than any one thing, there is the desire to be in the forefront of new developments and to inject a note of hope for better things to come.

When first in the House of Delegates one might feel intimidated by the protocol in being recognized to speak at reference committee hearings or on the floor of the house. The thought that you might embarrass yourself or your delegation lurks in the back of your mind. Nonetheless, the desire to put forth your feelings on a given point overshadows this.

As mentioned in my original article, the chairmen of the reference committees and speaker of the house help you in every way possible to get your questions or motions formulated. For the rest of my life, I will not forget the camaraderie and the sense of being a part of furthering health matters.

I fervently hope that more physicians will find a way to expand their own outlook and come to be a part of this worthy cause.

The past 12 years have shown that the AMA has energetically sought ways to represent all of medicine while continuing its excellent scientific reports and other endeavors to improve the health of America. We may not agree with all positions held by the AMA but we must agree that it is the one voice that speaks for medicine.

Stewart J. Petrie, M.D.

Branford

50 Years Ago

From *The Connecticut State Medical Journal*

February 1943

The Spa In America

THE use of mineral springs for medical purposes in America is a chapter in our annals which is not only of historical interest but one which seems destined to record a new development. There is good reason to believe that with the development of scientific knowledge of this medical regime which for 2,000 years has flourished in Europe our own spas may become important for us. Dr. Henry Sigerist[1] in an interesting essay on American Spas in Historical Perspective has shown how important were these therapeutic agencies during the 18th and 19th centuries, and offers some pertinent reasons for their decline in our own time. One of these is found in the fact that most of our more notable resorts became famous more for entertainment, such as horse racing and gambling, than they did for curative waters. Because of this development, these glamour spots sooner or later had to compete with similar resorts which although lacking waters were more accessible and more entertaining. The lush state which one of our celebrated spas engendered may be viewed in fine perspective in Miss Ferber's novel "Saratoga Trunk."

One reason for the popularity of the European spa has been that many of these establishments are state owned and are, therefore, accessible to the great mass of the people. In our country treatment at a spa seems to have been developed primarily for those in the high income groups, but even these people when possible liked to take their spa treatment at some highly developed European resort. Oskar Baudisch, Research Director at the Saratoga Spring Foundation, estimates that in 1930 100,000 Americans took treatment at European spas, spending there 100 million dollars.

In the opinion of Sigerist the scientific attitude toward spa treatment is to use natural curative forces on a basis of the rich clinical experience already available today and to promote energetically laboratory and clinical research. The most extensive and recent development of this kind has come through the establishment by the State of New York at Saratoga Springs of the first research institute of its kind on this continent. In this project the state has spent about 10 million dollars. This enterprise promises to be the beginning of a new health resort movement on a more scientific basis. In 1938 the American Medical Association appointed a Committee on American Health Resorts which will report on approved establishments and prepare a series of papers on the subject. At Saratoga Spa the research division has been particularly interested in enzyme like action or catalytic action of the waters and the effects which emphasize the significance of the so-called trace metals. These elements are studied by radioactive and isotopic means and are said to partake of the nature of "inorganic vitamins." A recent editorial[2] in commenting on this development states, "It would seem beyond doubt that this work will elevate natural mineral water research to the same level as vitamin and hormone investigation."

Perhaps Connecticut physicians will take added interest in this newer health resort development, recalling that our own history of the spa treatment of disease is not insignificant. As early as 1719 the Indians made the settlers at Stafford acquainted with the virtue of the springs, which later became a celebrated health resort. This is also an opportunity to recall that in 1766 to the town of Stafford came a young physician of Boston, Joseph Warren, who, having heard of the medicinal properties of the waters, purchased two tracts of land and later made plans for the building of a sanatorium. A hero's death at Bunker Hill prevented the fulfillment of a career and an enterprise which might have been significant in our State.

Through the newer scientific knowledge of the therapeutics of mineral waters it may be contemplated that health resorts in America in the future may play an important part in the treatment of disease. One fact is impressive, for twenty centuries, in spite of changing medical theories to explain the effect of medicinal waters, countless thousands of patients have found relief in their use.

REFERENCES

1. *Bulletin of the History of Medicine*, February 1942.
2. *Medical Times*, November 1942.

—Editorial from *The Connecticut State Medical Journal*, February 1943.

INQUIRE TODAY.

ENDORSED BY
THE CONNECTICUT STATE MEDICAL SOCIETY
—OFFERING—

- Hospital, Surgical and Major Medical Insurance
- Term Life Insurance
- Employee Benefit Package

—FEATURING—

- OFFICE OVERHEAD AND DISABILITY INSURANCE PACKAGE
  [Rates based on a 30 day waiting period for ages 30-39]
  $1500.00 weekly disability indemnity
  semi-annual premium is $528.15*
  $20,000.00 monthly overhead indemnity
  semi-annual premium is $561.00

* Optional Residual and COLA benefits available

AYR INSURANCE AGENCY

105 SANFORD STREET HAMDEN, CT 06514 288-4302

Biomedical Research—Our Moral Responsibility

Science has given to this generation the means of unlimited disaster or of unlimited progress. There will remain the greater task of directing knowledge lastingly towards the purpose of peace and human good. Sir Winston Churchill.

Every doctor who has gone through the rigors of medical school and residency training understands that most of the achievements to keep people healthy and to eradicate diseases were brought on by advances in biomedical research. From the days of Edward Jenner, the English physician who discovered that vaccination with cowpox gives protection from smallpox, through the days of Jonas Salk and Albert Sabin who invented dead-virus and live-virus vaccines against poliomyelitis, and to the present day of innumerable research projects including advanced gene therapy, new pharmaceuticals, stapling devices, and laparoscopic technology, medical and technological research has formed the foundation upon which our progress has been achieved. Imagine what would have happened to the health of the public if there were no inventions in the medical field and no advances in the basic biological sciences.

In these days of shrinking resources, the easy solution to trimming the budget is to cut back on the money spent on biomedical research. This is a terrible tragedy and legacy to leave behind for the future generations of our country. We have to convince the public and the government that a dollar spent on basic medical research today will give millions of dollars worth of benefits in the future. We have to fight for increasing amounts for our universities and other research institutions to proceed with this task and also to compete with the research projects of the foreign countries.

The other aspect of basic biomedical research involves the use of animal models. Using various animals ranging from the mouse to the chimpanzee has provided the researchers innumerable advantage in furthering the knowledge of science. Almost all individuals who are involved with this type of research activity are keenly aware that these animals should be treated humanely. There are strict protocols for maintaining and managing the animal laboratory. Recently this has been under attack by a group of extremists who would not hesitate to do anything to obstruct the ongoing research projects. This has to be stopped before it is too late. Thomas Hardy said in 1874: "A resolution to avoid an evil is seldom framed till the evil is so far advanced as to make avoidance impossible." That is why we as physicians have the moral responsibility to let the public know the advantages of medical research including the use of animal models. This is urgently needed.

In this regard, I would like the doctors of Connecticut to be informed about the organization "Connecticut United for Research Excellence" (CURE). The mission of CURE is " to advance biomedical research through public education, information, and support." The membership of CURE includes educational and research institutions, hospitals, voluntary health organizations, and professional societies. The advisory board of CURE includes representatives from the Connecticut State Medical Society.

The goals of CURE are the following: 1. Increase public awareness of the importance and benefits of biomedical research, and the process by which it is conducted. 2. Strengthen relationships between the individuals who conduct research, the beneficiaries of biomedical research, and the media to improve the public understanding of biomedical research. 3. Enhance support for research and interaction among the various interest groups involved in and benefiting from the research.

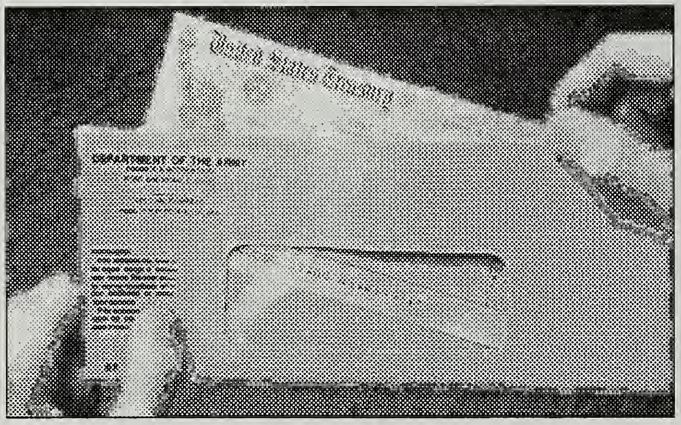
The Connecticut State Medical Society actively supports these goals. What can we as doctors do to facilitate this process? One thing we can easily do is distribute CURE's very attractive educational brochure "Pictures of Health." Complimentary copies for display in your waiting room are available by calling the CURE office at 294-3521 or by writing to CURE, P.O. Box 5048, Wallingford, CT 06492. Once again I urge all of you to talk to your patients regarding this subject. Once the knowledge and understanding of biomedical research is grasped by the public, we can overcome the obstacles and achieve true scientific freedom to make progress for the next century.

"Science is always simple and always profound. It is only the half-truths that are dangerous." George Bernard Shaw.

Sultan Ahamed, M.D.
President

ANESTHESIOLOGISTS AND SURGEONS: COULD YOU USE AN EXTRA $9,000?

If you're a resident in anesthesiology or surgery, the Army Reserve will pay you a yearly stipend which could total in excess of $9,000 in the Army Reserve's Specialized Training Assistance Program (STRAP).



You will have opportunities to continue your education and attend conferences, and we will be flexible about scheduling the time you serve. Your immediate commitment could be as little as two weeks a year, with a small added obligation later on.

Get a maximum amount of money for a minimum amount of service. Find out more by contacting an Army Reserve Medical Counselor.

Collect (203) 624-5325

ARMY RESERVE MEDICINE. BE ALL YOU CAN BE.®

Lyme Disease

The Lyme Care Center seeks qualified physicians throughout Connecticut to whom patients presenting symptoms of early, middle stage and chronic Lyme disease can be referred for treatment. Candidates for referrals must treat aggressively, appropriately, and compassionately. To those physicians who qualify for inclusion in our network, we offer a tremendous opportunity for practice enhancement. **Interested physicians should contact Joseph A. Hernandez, Managing Director.**

1-800-842-5248

HoMed/The Lyme Care Center
2371 Boston Post Road
Guilford, CT. 06437-2315

HoMed

THE LYME CARE CENTER℠

CALL FOR PAPERS

Members of the Connecticut State Medical Society reading papers before other organizations are requested to submit their papers to the JOURNAL for consideration by the Board of Editors for publication. Authors preparing manuscripts for submission to *Connecticut Medicine* should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office. Adherence to the instructions will prevent delays both in acceptance and in publication. Papers that have been set on a computer with foppy disk capabilities (3-1/2 or 5-1/4) should be submitted on disk along with the hardcopy andindication as to which wordprocessing program was used.

Please send them to:

Robert U. Massey, M.D.
Connecticut Medicine
160 St. Ronan Street
New Haven, CT 06511

REFLECTIONS ON MEDICINE

Managed Competition: The Rogue Wave of the Future?

ROBERT U. MASSEY, M.D.

LAST week an old friend, a retired physician, and I were talking over lunch. "Even though I've closed my office, I've had to keep my secretary," he said. "Her only job is to take care of my wife's and my Medicare and medigap insurance stuff. It's absolutely beyond me. How do others—not physicians like us—manage all this mess?"

To be sure, he has had a serious illness, but most of us in the Medicare crowd have more than one medical problem, or soon will have. In the past two months, following a visit to the doctor, we have received 18 medical or laboratory bills, twice that many incomprehensible insurance or Medicare documents, instructions, or forms, and half of them in error. For a while I was set to ignore them all, but then I realized that could get costly. It does little good to call the "if you have any questions" number; you wait on hold, listening to dreadful music for endless minutes, in despair, hang up, try again, and finally learn that the person you must speak to is "away from her desk," but will call you back. Maybe. Once there was a little grim humor from a clerk who was running out of patience: "Isn't it incredible," she said, "we put a man on the moon, but we sure don't know how to manage medical insurance. Don't tell my boss I said that!"

A general internist friend, a decade or so from retirement, is seriously considering getting out, running a travel agency, anything. "I work 12 hours a day just to keep up with the overhead, see my patients too briefly, and spend three or four hours every night catching up with the paper work." He is not alone. "Fully a third to as many as 50% of doctors are giving serious thought to whether they want to make a change," says Marilyn Moats Kennedy, a consultant with the American College of Physician Executives. "They are leaving direct delivery to become a medical director or working for a consulting firm.... They don't like the paperwork, and also the adversarial relationships and working long hours."[1]

From whatever point of view, patient, physician, employer, or politician, the system has crashed. My memories of doctoring in the 1940s and 1950s are those of good-natured colleagues who loved their work and looked after mostly satisfied patients. A selective memory perhaps, but I believe medical care was better then from almost every point of view, even though our new science and technology are an astonishment. If we have exchanged care that was not always effective for today's amazements, one of which is the medical corporation, I am not sure the trade-off has been worth it.

Now we have managed care, and we are about to have managed competition. The insurance companies, all 1,500 of them, will do the managing, mostly in their interests, and, except for the small, weak ones, they will not hurt one another, nor will they really compete or control costs; they will give only lip service to quality. They will profit handsomely while adding little of value to medical care, and that little will cost billions; we, doctors and patients alike, must finally drown in a raging sea of paper. Their only concept of management is to "intrude into health care decisions that ought to be made professionally," writes Arnold Relman, adding that he hopes the new administration "will consider alternative versions of the 'managed competition' idea that would put the responsibility for cost and quality control where it belongs—on physicians and their patients."[2]

As our conversation over lunch went along on its gloomy way, my physician friend predicted things would soon get so bad that some intemperate, ill-conceived scheme would be "slapped on us." A retired executive and hospital board member said, "The only answer is to cap the whole damned thing, and let the experts, the hospitals and the doctors, figure out how to make it work." He and Dr. Relman may have been thinking along similar lines!

REFERENCES

1. Newman L: Are younger physicians less unhappy in medicine? *Am Med News* 1993; 4 Jan., p. 15.
2. Relman AS: Controlling costs by "Managed Competition"—Would it work? *N Engl J Med* 1993; 328:133-5.

ROBERT U. MASSEY, M.D., Professor Emeritus, Division of Humanistic Studies in Medicine, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgement), provided that the are submitted in dupli–cate, signed by all authors, typewritten in double spacing, and do not exceed 1-1/2 pages of text (excluding references). They should not duplicate similar material being submitted or published elsewhere. Letters referring to a recent *Journal* article should be received within six weeks of the article's publication.

MALPRACTICE *vs* HUMANISM

To the Editor: In the October issue Dr. Ahamed ponders the dilemma of prolonging life when prospects for survival are unlikely and the quality of life attainable is gruesome.

As elements contributing to the problem he mentioned professional pride, fear of malpractice, and a variciousness.

However, of these three possibilities only one is an inescapable and ubiquitous fact. I refer to the specter of malpractice.

Until it is controlled, our attempts at inculcating the humanism which the author advises will be futile.

Edward J. Volpintesta, M.D.
Bethel

SELF-RAPE IS PARTIAL SUICIDE

To the Editor: Drug addiction is a disease of self-rape. It robs the victim of human character. To be human is to have integrity; there is an expectation of decency and honesty. But that imperative compulsion to consume a dose of drug overwhelms every trace of civilized personality.

The death of an inner quality has occurred, the horrendous deception is that the external appearance does not change enough to be easily recognizable. And in a certain Asiatic country this is dealt with by utilizing euthanasia.

Addicts are everywhere, some operate big trucks and accidents occur. With our permissive and lax society, the accidents continue. The benefits of society's protection should be weighted in favor of the self-disciplined: no one should have to die from a highway accident because some who drink still want to drive.

Harmony demands balance and proportion, and a safe society is a basic essential. One man's liberty should not be the death of another. Adequate regulation is required for the general safety! Self-rape is partial suicide.

Sidney Vernon, M.D.
Willimantic

RBRVS IN RURAL CONNECTICUT

To the Editor: The combination of the RBRVS institution and the Adjusted Historic Payment Base has produced gross discrepancies in the way physicians are paid across the state of Connecticut. The effect of this is to discourage new physician recruitment in rural areas.

To give a few examples:

CPT Code 1992
Difference

42140	Area 2	Area 4	
(Uvulectomy)	$2,172	$102	2,000%
31255	Area 2	Area 4	
(Ethmoidectomy)	$1,413	$629	125%
67916	Area 3	Area 4	
(Ectropian Rep)	$725	$478	50%
67228	Area 2	Area 4	
(Photocoag)	$780	$608	28%

These figures have brought HCFA's credibility level with my colleagues to an all time low.

It is certainly the government's desire to help distribute the medical army to its people so that even those in rural districts have the benefit of adequate medical care. This was after all the intent of the Public Health Service so that physician shortage areas could be adequately served. The current system, however, rewards physicians for practicing in high density population areas by reimbursing them for expenses which they themselves incurred by living in more lucrative areas. Doctors wishing to practice in such locations do so by choice and it is not up to the federal government to help them out here. Even the final RBRVS rules and formulas will discourage any physician from practicing in areas not at least equally reimbursed. A minority of phy–sicians feel the rural designations should have geographic practice cost index's (GPCI's) higher than urban ones in the inverse of the ratio proposed so that physician shortage areas can be made more desirable.

Over the next five years "Area 4" hospitals and phy–sicians will have an insurmountable task of recruitment. There have already been several excellent candidates to the Windham/Putnam area in different specialties who have turned down positions here simply on the basis of inade–quate compensation when compared to even the neighboring counties. The Connecticut State Medical Society urged HCFA to institute a simple statewide fee schedule about one year ago and HCFA replied that they would be glad to comply if all the doctors in the state agreed to this. Clearly, physicians in areas 1 and 2 would never do this and it was wrong of HCFA to suggest it. Changes must be mandated and fair so that all patients will benefit in the end.

The elderly of northeastern Connecticut will be having increasing problems with health care access due to the shortage of doctors here. HCFA has placed roadblocks in our attempt to recruit physicians to our area. We understand that HCFA does not intend to make further changes in the locality system until 1996, but this is a very important issue that warrants attention immediately.

Steven H. Stein, M.D.

President, Windham County Medical Association

Willimantic

BIRTH OF A NEW HEALTH CARE SYSTEM

To the Editor: The profession of medicine is about to undergo a passage, a midlife crisis, a transition, a change of life. What this change will be is unclear, whether we will be able to influence the change is uncertain, how we could structure the change (if we could) is unknown even to us.

But if we are to have any influence over our future, we must examine our soul. What are our priorities? Why did we train for this career? Why do we undertake the lengthy, exhausting process which delivered us to this demanding profession?

Until we know who we are, until we define our values, we cannot defend them.

Is it money which motivates us? If so, then any restructuring of the health care delivery system is acceptable so long as our income does not change.

Many of us saw medicine as an autonomous profession, imagined its practitioners as unencumbered by "bosses" or petty rules and regulations, time clocks, and bureaucracy. The poor souls for whom this is the highest priority must surely be in purgatory today and would accept changes that hold out the promise of a major decrease in the "hassle factor."

For others, our profession held out the primary prospect of being a healer, of entering the relationship between doctor and patient. To these physicians the major threat today is the determined effort one can see coming to interpose a phalanx of computers and technicians between doctor and patient.

Which spectre frightens us most? Which change is to be fought at all costs? Until we know, we cannot effectively participate in the process of forging a health care system which will be able to increase access while controlling costs. These goals have clearly been ratified by the American people, even as other goals remain clouded.

If we are to attend the birth of a new health care system, we first need to explore our souls.

Steven Wolfson, M.D.

New Haven

YOCON®
YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of YOCON® 1/12 gr. 5.4mg in bottles of 100's NDC 53159-001-01, 1000's NDC 53159-001-10 and Blister-Paks of 30's NDC 53159-001-30

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85

NDC 53159-001-30
YOCON®
Yohimbine Hydrochloride
SEALED FOR YOUR PROTECTION
NDC 53159-001-01
YOCON®
YOHIMBINE HYDROCHLORIDE
5.4 mg. (1/12 grain) per tablet
100 TABLETS
manufactured for
PALISADES PHARMACEUTICALS, INC.
Tenafly, New Jersey 07670
NDC 53159-001-10
YOCON®
YOHIMBINE HYDROCHLORIDE
5.4 mg. (1/12 grain) per tablet
1000 TABLETS
DOSAGE: 1 tablet 3 times daily, or as directed by a physician. See product literature for complete information.
CAUTION: Federal U.S. law prohibits dispensing without prescription.
Preserve in well-closed container.
PACKAGE NOT CHILD RESISTANT
Lot No.
Exp.
manufactured for
PALISADES PHARMACEUTICALS, INC.
Tenafly, New Jersey 07670
by:
Bowman Pharmaceuticals, Inc.
Canton, Ohio 44702

Available at pharmacies nationwide
PALISADES
PHARMACEUTICALS, INC.
219 County Road
Tenafly, New Jersey 07670
(201) 569-8502
(800) 237-9083

This Month's Reading in Review

TIMOTHY B. NORBECK

- A recent study by two economists at the State University of New York in Buffalo, published in the journal of *Applied Economics*, found that when other sources of lawyers' incomes dry up or as competition increases, attorneys tend to pursue more medical malpractice claims...."Specifically," the study indicated, "a 10% increase in lawyers' excess time drives up the number of medical malpractice claims by 4%."
- "We do not have global patients. We take care of patients one at a time, and I don't want a bureaucrat in my examining room when I'm taking care of a patient telling me what I can and cannot do." Ray Scalettar, M.D., Chairman, AMA Board of Trustees, at a recent press conference.
- The Coors brewing company is dramatically lowering employee health care costs with an effective wellness program....It is one of a few U.S. companies offering on-site mammograms (for only $5) and has spent almost $600,000 on screenings....An early detection case has cost them $11,000 compared to $140,000 for a late detection case....Under health reform, other companies might well be asked to provide wellness programs such as the one implemented at Coors.
- The Coors wellness program has saved the company overall some $1.9 million every year by decreasing medical costs, reducing sick leave and increasing productivity....For every dollar spent on wellness, Coors sees a return of $6.15.
- 1992 is the first year that participating physicians in Medicare (52.2%) represent a majority of those in the program (48% in CT)....By comparison, only 23.7% of medical suppliers (such as those providing ambulance services or durable medical equipment) are considered participators....Assigned Medicare claims represented 87.6% of Part B's covered charges nationally while the figure was 89% in Connecticut.
- Another demographic reason for rising health care costs: those 85 and over are the fastest growing segment of the population and have expanded by 232% since 1960 compared to 39% for the population as a whole....In addition, the number of centenarians has doubled from 18,000 in 1982 to 36,000 today.
- On average, someone over 85 years of age has health care costs six times higher than someone under 65....That age group, comprising 3 million in 1990, is expected to double to 6.1 million in 2010....This is an important factor in the health care cost debate.
- AARP's power is certain to increase considerably over the next two decades....Their present membership stands at 34 million members, and the national population aged 50 and older is projected to grow from 65 million today to 97 million by 2010....Such growth represents an increase of 49% over that period while the population under age 50 is expected to increase by only 1%.
- Overall, 88.8% of Medicare's annual revenue now comes from people who are under 65 years of age (in payroll taxes, income taxes, and trust-fund interest)....The balance of 11.2% comes from those 65 and older (in premiums).
- As reported recently in the *New York Times*, the German Parliament has approved a package of health care changes intended to curb the surging costs of medical and dental care by making patients pay more and forcing doctors, dentists, hospitals and drug companies to cut prices....The measures took effect January 1, 1993, and were vigorously opposed by the health care industry....In fact, they prompted Germany's 43,000 dentists to stage a one-day nationwide strike this past summer when they were first announced.
- *Only in America:* The *Wall Street Journal* reported that Brown University (Providence, RI) spent more than two years (plus $35,000 on outside lawyers in addition to its in-house lawyer) fighting a lawsuit over an injury incurred by a student from a soap tray in a dormitory shower stall. In the case, a female student sued Brown for $700,000 because she cut her upper arm on a broken soap tray while showering with her boyfriend. A federal court jury, after hearing a school janitor testify that the soap tray was intact before the couple showered, found in favor of the university.

TIMOTHY B. NORBECK, Executive Director, Connecticut State Medical Society.

Book Review

Medicine: The Art of Healing
By Sherwin B. Nuland, M.D., 115 pp., illustrated. New York, N. Y., Hugh Lauter Levin Associates, Inc., 1992. ISBN 0-88363-292-6.

My introduction to history came through the movies; without pictures—the making of images in the mind—how can you imagine or understand history, or even be interested in it? Who wants to know about the economic plight of the French wine and silkworm industry in the 19th-century, or about the German dye industry and its boost to the development of hematology, bacteriology, and cellular pathology by the selective affinity of protoplasm for analine dyes? "Louis Pasteur" and "The Magic Bullet" were only movies, yet no one who saw them would likely forget the great French chemist and his rabies vaccine, or Paul Ehrlich's feverish search for "606" or salvarsan.

Last week a friend, a businessman, said as we were leaving a restaurant, "Hey, I just got a great book as a gift—it's full of pictures about the history of medicine; you'd love it!" When he described his gift I knew he had been looking at the pictures and reading the text of Dr. Nuland's book, *Medicine: The Art of Healing*, a book the author insists in the Introduction "is not a history of medicine."

Yet when I first got my hands on it, I thought to myself, "This is the text we should be using in our seminar for medical students in medical history." In a much too brief introduction to the history of medicine, what will they remember of the four humors or the Paris school, of Pierre Louis's numerical system, of William Osler, William Stewart Halstead, or William Henry Welch? It is impossible in half a semester to be complete, to explore all the angles; only pictures, and sometimes artifacts, can bring us close to the past, enabling us to imagine it, in the words of the eminent 19th-century historian von Ranke, *wie es eigentlich gewesen*, "as is really was."

Dr. Nuland has assembled a collection of 48 color-plates beginning with a 12th-century Byzantine manuscript of the Oath of Hippocrates and ending with a 1975 tapestry made from a late 1930s photograph of the Canadian surgeon Norman Bethune operating under primitive conditions at a guerilla base in China. These pictures are those that especially appealed to the author, each one selected because he felt it was important, he had enjoyed looking at it, and he had profited from studying it. His readers will mostly agree, and will especially enjoy reading his account of the pictures, the artists, and the physicians, their significance in medical history, and how they portray in objective detail the nature of some aspect of medicine and society in their own times. Despite the author's denial, this collection comprises in memorable form and color the sweep of western medical history from the 12th to the 20th-century.

Because it does not overwhelm it cannot provide an "encyclopedic reference work for those who would delve into details," nor does it explore all the ways in which art has served medicine and medicine art. The portraits of Pasteur and Ehrlich are not part of the collection! But color-plates and text together make a fine introduction to the history of medicine, that most human of all sciences.

Medicine: The Art of Healing is a handsome quarto volume; the plates are well reproduced and the place is given where the originals may be seen. Some are familiar, but many will never have been seen before by most readers. It by no means duplicates other pictorial history of medicine productions.

Robert U. Massey, M.D.

You'll love working with our locum tenens physicians and allied health care professionals.

WE GUARANTEE IT.

CompHealth has thoroughly credentialed physicians and allied health care providers from more than 40 fields of specialization available to provide locum tenens, or temporary, staffing assistance when and where you need it.

Plus, we have the standards and experience to guarantee your satisfaction each time we place a member of our medical staff in your practice or facility. It's the closest thing you'll find to a risk-free way to cover for absent staff members, "try out" a potential new recruit, or take care of your patients while you search for a new full-time associate.

Call us today to arrange for quality locum tenens coverage, or to discuss your permanent recruiting needs.

CompHealth
COMPREHENSIVE HEALTH CARE STAFFING

1-800-453-3030
Salt Lake City ■ Atlanta ■ Grand Rapids, Mich.

CSMS PHYSICIAN PLACEMENT SERVICE

The Society maintains the Physician Placement Service as a *free* service to the medical profession, hospitals, and communities in Connecticut.

***Opportunities* should be typed, double-spaced copy on letterhead and submitted to CSMS, Physician Placement Service, 160 St. Ronan Street, New Haven, CT 06511 (203) 865-0587 or fax to (203) 865-4997. These will be published as space permits and will be distributed to physicians making inquiries of such *opportunities*.**

***Physicians wishing to locate* in Connecticut may call the office requesting opportunities in their specialty. Also, candidates are invited to submit a resume to be kept on file with the Society. An announcement of a physician's availability will be published in two issues of *Connecticut Medicine* as space permits.**

Listing of physicians in the Placement Service does not in any way represent certification by the Society. Investigation of credentials and experience is the responsibility of those seeking applicants for positions.

Announcements on the Physician Placement Service page under Classified Advertising are charged at the regular Classified Advertising rate.

OPPORTUNITIES FOR PRACTICE

AMBULATORY CARE

Full time or part time positions available in ambulatory care offices in Central Connecticut. No on-call duty. Pleasant conditions and staff. Moderate volume practices. Please apply to Medical Director, Newington Medical Care Center, 955 Main Street, Newington, CT 06111.

Full-time or part-time position available for physician (Family Practice, Internist, or Emergency Medicine) in established Walk-In Center. Highly competitive salary and benefits; very pleasant working conditions; located on beautiful Connecticut shoreline in family community. Please send CV or call: Harry Ardolino, M.D., Coastline Medical Center, 9 Business Park Drive, Branford, CT 06405. Phone: (203) 481-0818.

EMERGENCY MEDICINE

Busy walk-in center seeking full-time or part-time physician, requires general medicine, minor-surgery and urgent-care treatment experience. Wonderful staff, great community, good salary and benefits. Contact: Dr. T. Kandell/Dr. R. Falit, Med-Help Medical Center, P.O. Box 1120, Bristol, CT 06010. Telephone: (203) 584-8900.

Emergency Physician - Full time position for board prepared, experienced emergency unit physician at an 85 bed acute hospital in central Connecticut. Current Connecticut license required. Includes involvement in Occupational Health program. Competitive salary and excellent benefit package. Call John Scarfo, M.D. @(203) 276-5210 or send CV to John Scarfo, M.D. Chairman Emergency Medicine, Bradley Memorial Hospital, 81 Meriden Avenue, Southington, CT 06489. EEO/M/F/D/V.

FAMILY PRACTICE

Southwestern Connecticut-Family Practice-Ideal opportunity for motivated and energetic BC/BE Family Practitioner to join solo provider in rapidly growing private Family Practice. If you have been thinking about entering into a solo or private Family Practice this is a unique situation. The groundwork has been laid and with the right individual(s) this practice will develop into a quality Family Practice Group. Open and flexible to discussions regarding the position and expansion of the practice. Please contact via telephone and send C.V. to: Alan T. Falkoff, M.D., 838 High Ridge Road, Stamford, CT 06905. (203) 322-7070.

Two Family Practice group looking for 3rd associate. Traditional family practice (no OB) with good mixture of patients. We admit patients to local hospital that trains family practice residents. Please contact Susanna Thomas, M.D. Telephone: (203) 388-1495.

General Practice for Sale.— Retiring in April 1993. Center of Wallingford CT. Certified Medical Assistant available to continue (billing, computer EKG lab work). Convalescent homes nearby. Yale New Haven and local hospitals 15 minutes. Contact Eugen Krc, M.D. at (203) 265-3300.

INTERNAL MEDICINE

Stratford internist group seeks generalist or subspecialist to join established practice. Flexible working schedule and part-time hours are possible. Please respond to CSMS c/o IM/WC.

Full-time or part-time position available for physician (Family Practice, Internist, or Emergency Medicine) in established Walk-In Center. Highly competitive salary and benefits; very pleasant working conditions; located on beautiful Connecticut shoreline in family community. Please send CV or call: Harry Ardolino, M.D., Coastline Medical Center, 9 Business Park Drive, Branford, CT 06405. Phone: (203) 481-0818.

The Postoperative Incidence of Small Bowel Obstruction Following Standard, Open Appendectomy and Cholecystectomy: A Six-Year Retrospective Cohort Study at Yale-New Haven Hospital

ROSS I.S. ZBAR, M.D., WILLIAM B. CREDE, M.D., CHARLES F. MCKHANN, M.D.,
AND JAMES F. JEKEL, M.D., M.P.H.

ABSTRACT—The incidence of postoperative small bowel obstruction (SBO) after standard, open appendectomy and cholecystectomy was calculated during a six-year period at a university medical center hospital, which is the larger of two local, community hospitals. A cohort of 567 patients who underwent either a standard, open appendectomy or cholecystectomy from 1 October 1985 through 30 September 1986 was assembled. Of these patients, 182 (32.1%) were readmitted to the hospital prior to 1 October 1991 and thereby received follow-up. The time-related incidence of readmission to the hospital with a specific diagnosis of SBO as estimated by the Kaplan-Meier method was tabulated. This analysis revealed the following incidence rates of postoperative SBO: 10.7% following appendectomy during 64 months of follow-up (n=41) and 6.4% following cholecystectomy during 67 months (n=141). The Kaplan-Meier product-limit incidence of postoperative SBO was significantly different for standard appendectomy versus standard cholecystectomy (Breslow-Cox *P* value < 0.0277). This implies that the anatomical position and/or the likelihood of perioperative infection associated with open, abdominal surgery plays a significant role in subsequent adhesion formation and development of SBO. These data may be compared to laparoscopic techniques in future studies.

Introduction

POSTOPERATIVE small bowel obstruction (SBO) secondary to adhesive bands is a well described entity with a complex pathophysiology. Following surgery in the abdominal cavity, vascular growth occurs towards ischemic peritoneal tissue. When the ischemic crisis has passed, the vascular bridges are reabsorbed, leaving a fibrous matrix. Over time, this matrix may contract, resulting in external occlusion of the gastrointestinal lumen.[1] Other factors associated with postoperative adhesion formation include foreign body granulomata, chemical irritation to the peritoneum, and thermal injury.[2,3] Although many previous studies have examined the operations most likely to precede SBO, few have sought to measure the actual incidence of SBO following common abdominal procedures.[4-12]

A recent prospective, regional study reported an incidence of 39.9 cases of SBO per 100,000 population per year.[13] However, this study observed all patients with obstruction; including those with postoperative adhesions, malignancies, and strangulated hernias. Moreover, the calculated figure was a *heterodemic* ratio, which was generated by including all those patients with an obstructing event in the numerator and using the size of the regional population in the denominator. In actuality, since not all patients in the denominator underwent surgery and were therefore not at risk for postoperative SBO, this incidence rate is inappropriate.

Very few studies exist which quantify the incidence of postoperative SBO following abdominal operations. Both Lewis et al[14] and Owens and Hamit[15] investigated the incidence of postoperative obstruction following appendectomy, with their results cited in a recent text.[3] The quoted incidence of 0.2% obstruction following appen-

---

ROSS I.S. ZBAR, M.D., Department of Surgery, Yale University School of Medicine, New Haven; WILLIAM B. CREDE, M.D., Quality Improvement Support Service, Yale-New Haven Hospital; CHARLES F. MCKHANN, M.D., Department of Surgery, Yale University School of Medicine, New Haven; JAMES F. JEKEL, M.D., M.P.H., Department of Epidemiology and Public Health.

Reprint Requests: Ross I.S. Zbar, M.D., Department of Surgery, Lenox Hill Hospital, 100 E 77th Street, NY, NY 10021.

dectomy from Lewis et al represented only early postoperative SBO in patients who had not yet been discharged from the hospital. Owens and Hamit, incorporating patient follow-up in their study, arrived at an incidence of 2.9% obstruction following appendectomy. However, this study was limited to a population older than 65 years of age and failed to account for either patient loss to follow-up or the varying duration of observation. Melody[16] cited an incidence of 2.8% obstruction following total abdominal hysterectomy from a personal series of patients. Unfortunately, Melody, like Owens and Hamit, failed to account for either loss to follow-up or variations in duration of observation. Sannella[17] reported an incidence of postoperative SBO following abdominoperineal resection of 6.8% over 21 years of follow-up. When recurrent carcinoma was included as a cause of postoperative obstruction, the incidence increased to 10.8%. Although Sannella used follow-up data to calculate an incidence of postoperative SBO, he failed to account for variations in duration of patient observation.

To obtain information concerning the time-related incidence of SBO after standard appendectomy and cholecystectomy, the present study utilizes a retrospective, longitudinal cohort design.[18] Calculation of incidence rates for postoperative SBO is undertaken using the Kaplan-Meier product-limit method, which adjusts the incidence ratio to account for the various durations of follow-up and the losses to follow-up experienced by each patient within the observed cohort.[19] This study design is particularly powerful in that it is *homodemic*,[18] where each patient in the cohort who is at risk for SBO is accounted for through the use of follow-up data. This homodemic design eliminates any speculation about patient outcome following surgery. Also, even with low overall incidence, the Kaplan-Meier product-limit calculates an accurate estimation of the rate.

Patients and Methods

Study Population.—A cohort of surgical patients from Yale-New Haven Hospital (Y-NHH) was assembled which included all those patients who underwent either standard, open, primary appendectomy or cholecystectomy during fiscal year 1986 (1 October 1985 through 30 September 1986), and who were discharged from the hospital. These patients were identified through a computer search of a data base in the Clinical Information Service (CIS) at Y-NHH, which included all primary operative procedures performed and coded according to the *International Classification of Diseases —Ninth Revision* (ICD-9)[20] code. Patient follow-up data were obtained by tracking each patient in the surgical cohort through the use of computerized admitting diagnosis codes in the CIS database, from fiscal year 1986 through fiscal year 1991 and noting when, and why, a patient was readmitted to Y-NHH. Patients who came back to Y-NHH with any subsequent diagnosis or procedure were considered to be under observation up to their most recent hospital admission date, which was used to calculate the duration of follow-up. Those patients who were never readmitted to Y-NHH, for any reason after the index abdominal procedure, were considered to be lost to follow-up after discharge.

The complete medical records of all cohort patients with an admitting diagnosis code of SBO were then individually reviewed. Patients who lacked a discharge diagnosis of SBO, despite an admitting diagnosis of SBO, were not considered obstructed secondary to postoperative adhesions, because the admitting diagnosis was discovered to be in error. Additionally, cohort patients with SBO secondary to cancer, incarcerated inguinal hernia, or mesenteric vascular disease were not considered SBO outcomes for this study. Patients with either the admitting or discharge diagnosis of partial SBO, intermittent SBO, pseudo-obstruction (Ogilvie's syndrome), large bowel obstruction, fecal impaction, paralytic ileus, or abdominal pain were not considered to have the outcome event. No patients were rejected from this study on the basis of age or sex, but patients with portions of their medical records missing from the period under observation were disqualified.

The admitting diagnosis of SBO was based upon a combination of history (pain, nausea. vomiting, obstipation), physical examination (abnormal bowel sounds, abdominal distention, mass), and abdominal x-ray findings (dilated loops of small bowel, air-fluid levels). Not every patient had all of the signs and symptoms of SBO; however, each patient had radiologic evidence of SBO in combination with compatible history and physical examination findings.

Statistical Analysis.—The Kaplan-Meier product-limit method[19] was used to calculate the cumulative probability of an obstructive event following either appendectomy or cholecystectomy. This technique calculates incidence curves over time by using follow-up data from all individuals in the cohort, regardless of duration of follow-up. A statistical comparison of Kaplan-Meier incidence curves over time was performed using the Breslow-Cox test of significance (from BMDP Statistical Software, Inc.).

Results

Of the 567 patients in the postoperative cohort, 182 (32.1%) were subsequently readmitted to Y-NHH and, therefore, had follow-up data. Of these patients, six (3.3%) returned to Y-NHH with SBO which met the study criteria.

Appendectomy.—A total of 179 patients underwent primary appendectomy during fiscal year 1986, among whom 41 persons (22.9%) returned to Y-NHH for subsequent admissions. Of the 41 followed patients, three

(7.3%) developed SBO. This resulted in an adjusted Kaplan-Meier incidence rate of 10.7% during 64 months of follow-up (Table 1 and Fig. 1). Of the three obstructed patients, two (66.7%) had had perforated appendices at the time of index procedure.

Cholecystectomy.—A total of 388 patients underwent total cholecystectomy during fiscal year 1986, among whom 141 persons (36.3%) returned to Y-NHH for subsequent admissions. Of these 141 patients, three (2.1%) developed SBO. This resulted in an adjusted Kaplan-Meier incidence rate of 6.4% during 67 months of follow-up (Table 1 and Fig. 1). Of the three obstructed patients, one (33.3%) had had a perforated gallbladder at the time of index procedure.

Statistical Analysis of Kaplan-Meier Incidence Curves.—When the Kaplan-Meier incidence curves for postoperative SBO are compared between the appendectomy and cholecystectomy cohorts, a Breslow-Cox P value < 0.0277 was obtained (Fig. 1).

Comment

This study represents the first time, as far as we could determine, that the Kaplan-Meier product-limit analysis has been applied to the study of postoperative SBO. By retrospectively defining a postoperative surgical cohort, following the group forward in time, and observing for SBO, the life table analysis generated incidence curves. These Kaplan-Meier product-limit incidence curves take into account the relative duration of follow-up for each patient. This approach examined an old issue and revealed new information. Previous studies have been mainly descriptive in nature, usually only characterizing the obstructed group and failing to look at the entire population at risk. Furthermore, those previous studies which quoted incidence rates, when examined closely for statistical soundness, failed to account for patient loss to follow-up and varying durations of patient observation.

The value of the life table method is demonstrated by analyzing the data obtained in the present study using the techniques of previous studies. For example, there were 179 appendectomies performed in fiscal year 1986 at Y-NHH of which 41 patients returned for follow-up and three developed SBO over the ensuing six years (Table 1). Using heterodemic epidemiological techniques, this means that for appendectomy, there were three post-appendectomy SBOs per 350,000 population in the New Haven region over six years, or 0.00086% of population per year. This is a poorly defined figure because not every person in the denominator is at-risk for postappendectomy SBO, nor is there any accounting for loss to follow-up.

A more refined statistic, although still inadequate, is an incidence of three SBOs per 179 appendectomies performed, or 1.7% over six years. Although this figure attempts to account for only those patients at risk for post-appendectomy SBO, it is still imprecise because it assumes that all 179 appendectomy patients are still alive and would have returned exclusively to Y-NHH for treatment of obstruction, if it had occurred. This is obviously an invalid assumption, but it does set the minimum possible value of postoperative SBO for this cohort

A still better estimate of the incidence of SBO after appendectomy, which attempts to take follow-up into account, is an incidence of three SBOs per 41 observed patients over six years, or 7.3%. This value actually represents the percentage of observed patients who become obstructed; however, it does not account for varying duration of follow-up. Using Kaplan-Meier product-limit analysis, the best estimate of the incidence of obstruction, adjusted for duration of follow-up after appendectomy, is 10.7% during 5.3 years of follow-up, or about 2% per year of follow-up.

Cholecystectomy patients had an incidence of SBO of 6.4% during 67 months of follow-up, or about 1% per year of follow-up. This obstruction rate was significantly

Table 1

Incidence of SBO for Various Abdominal Procedures During Follow-Up (FU) Period

Procedure	*Total # of procedures performed in fiscal year 1986*	*# of pts returning for FU after procedure (%)*	*Total months of follow-up*	*# of pts with FU who then developed SBO (%)*	*Kaplan-Meier adjusted incidence over FU time*
Appendectomy	179	41 (22.9%)	64	3 (7.3%)	10.7%
Cholecystectomy	388	141 (36.3%)	67	3 (2.1%)	6.4%

lower than that of appendectomy ($P < 0.0277$). This is a reasonable finding, because anatomically, the field of dissection required to reach the gallbladder and its fossa involves less direct manipulation of bowel, and there is usually less preoperative inflammation occurring with cholecystitis than with appendicitis. The anatomical topography of the small bowel and its location during postoperative healing surely plays an additional role.

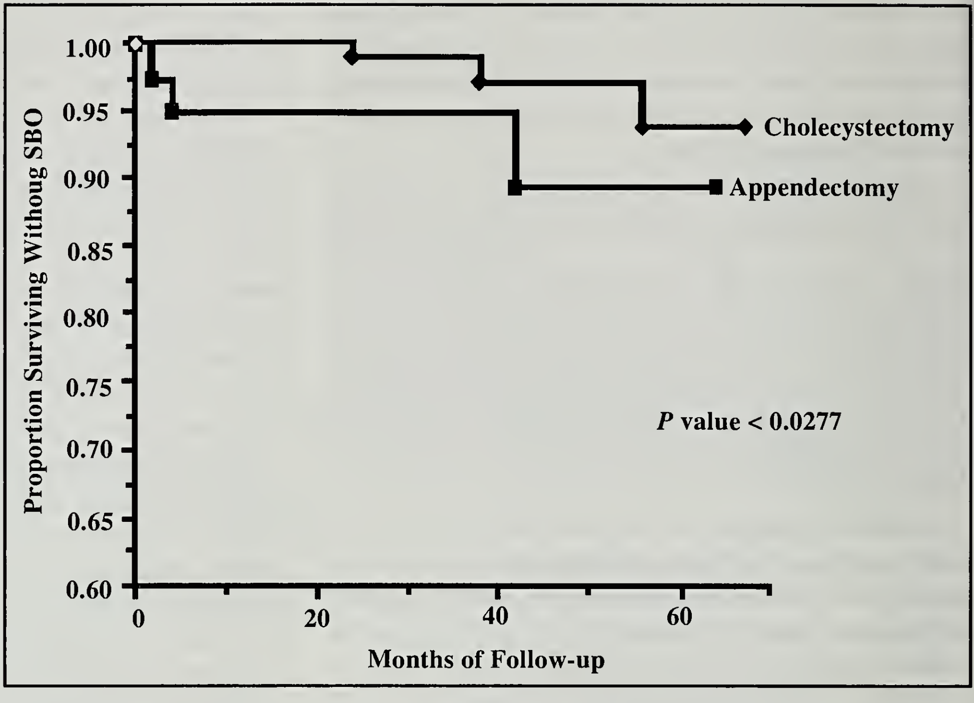


Figure 1
The Kaplan-Meier product-limit estimation of the time-related survival of cohort patients without an obstructive event following either appendectomy or cholecystectomy.

Surgeons may initially be surprised that the incidence figures for SBO following appendectomy and cholecystectomy were as high as tabulated in the present study. However, an accurate incidence figure requires careful research design and statistical analysis; rates derived from personal experience, without defined cohorts, chart review, and follow-up, are subject to bias.

One assumption used in calculating the Kaplan-Meier product-limit was that those patients who were subsequently readmitted to Y-NHH were dedicated users of the hospital, until they were lost to follow-up. Follow-up data were based on most recent hospital admission data and discharge diagnoses. This is believed to be a valid assumption, because those postoperative cohort patients who returned to Y-NHH demonstrated a preference for this hospital. Additionally, Y-NHH is the larger of two community hospitals in the New Haven area (excluding a Veterans Administration hospital).

It is possible that a few patients may have undergone their index primary operation at Y-NHH, had an obstructing event treated at an outside hospital, and then returned to Y-NHH for treatment of another ailment. This subgroup of cohort patients would not have been tabulated in the obstructed group, but would have been counted as receiving follow-up which was free of obstruction. Despite subsequent follow-up, there would be no way to know of their prior obstruction, and this might lead to a small underestimation of the incidence.

Some might argue that basing Kaplan-Meier product-limit analysis on follow-up data obtained from hospital readmissions skews the analysis towards a sicker cohort of patients. However, simple mechanical SBO is an independent event which occurs only as a result of adhesion formation and subsequent band contraction. Thus other medical problems requiring hospital admission should not select out a biased population with regard to the incidence of simple, mechanical SBO. This is an important assumption, for if the calculated incidence rates of postoperative obstruction are to be applied to the general, postoperative population, all patients lost to follow-up must possess a similar underlying incidence of SBO to the population which received follow-up. If, on the other hand, the population lost to follow-up experienced a lower rate of simple, mechanical SBO than the population which received follow-up, then the calculated incidence rates of SBO in the present study represent maximum values.

In the present study, an index abdominal procedure during a one-year catchment period was used as the baseline event for calculating the life-table incidence of postoperative SBO. Future studies should observe a postoperative cohort of patients without any other past surgical history, besides that of the index procedure. These results could then be compared to those obtained in the present study, in order to assess differences, if any, in the rate of obstruction between one-time and multiply operated abdomens.

The incidence rate of SBO over time following appendectomy and cholecystectomy was calculated in this study. Thus, for example, this study now permits surgeons to inform their patients that the 64-month risk for SBO after appendectomy is approximately 10.7%. However, data beyond this time period do not exist. Although the incidence of obstruction appears to be a front-loaded risk (Fig. 1), with more obstructing events occurring earlier, the fact that the incidence curves plateau between 40 and 55 months does not mean that obstructions will no longer occur beyond that point in time. Incidence rates cannot be extrapolated beyond the duration of follow-up because the rate of obstruction may not be constant over time.

Perforation of an abdominal viscus appears to increase the risk for subsequent SBO. This is presumably mediated by an increase in adhesion formation within a contaminated peritoneal cavity.[21,22] Two of three obstructed patients who had undergone appendectomy had had a perforated appendix preoperatively, and one of three obstructed patients who had undergone cholecystectomy had had a perforated gallbladder preoperatively. Unfortunately, the CIS database did not contain information regarding the status of each patient's abdomen at the time of index procedure. To settle this issue, future studies should perform life-table analyses on patients with and without gross peritoneal contamination and compare the resulting incidence rates of obstruction. Additionally, life-table analysis should be used to examine the postoperative incidence of SBO after incidental enterotomies.

Examination of the time-related incidence of SBO following common abdominal operations is of special interest as laparoscopic surgery becomes a popular method of entering the abdomen.[23] Studies have demonstrated that laparoscopic surgery is associated with less patient discomfort, shorter hospital stays, and faster return to preoperative activity levels.[24,25] It is not clear, however, whether or not laparoscopic surgery carries a decreased risk for postoperative adhesion formation and subsequent SBO. This study represents a new approach to the calculation of the incidence of SBO secondary to adhesion formation for two standard, open abdominal procedures, and therefore, it may serve as a comparison for future studies of laparoscopic adhesion formation and obstruction.

The authors wish to thank Chris Hilton for his computer programming expertise. We also thank Ronald R. Salem, M.D., and David I. Soybel, M.D., for their constructive commentary.

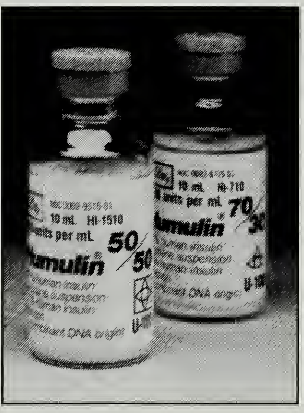
REFERENCES

1. Ellis H: The cause and prevention of postoperative intraperitoneal adhesions. *Surg Gynecol Obstet* 1971; 133:497-511.
2. Myllarniemi H: Foreign material in adhesion formation after abdominal surgery. *Acta Chir Scand* 1967; (Suppl) 377:1-48.
3. Welch JP: *Bowel Obstruction.* Philadelphia: W.B. Saunders Company; 1990.
4. Bevan PG: Adhesive obstruction. *Ann R Coll Surg Engl* 1984; 66:164-9.
5. Eliason EL and Welty RF: A ten-year survey of intestinal obstruction. *Ann Surg* 1947; 125:57-65.
6. Hofstetter SR: Acute adhesive obstruction of the small intestine. *Surg Gynecol Obstet* 1981; 152:141-4.
7. Laws HL and Aldrete JS: Small bowel obstruction: A review of 465 cases. *South Med J* 1976; 69:733-4.
8. Nemir P: Intestinal obstruction: Ten-year statistical survey at the hospital of the University of Pennsylvania. *Ann Surg* 1952; 135: 367-75.
9. Perry JF, Smith GA, and Yonehiro EG: Intestinal obstruction caused by adhesions. *Ann Surg* 1955; 142:810-6.
10. Playforth RH, Holloway JB, and Griffen WO: Mechanical small bowel obstruction: A plea for earlier surgical intervention. *Ann Surg* 1970; 171:783-7.
11. Quan SHQ and Stearns MW: Early postoperative intestinal obstruction and postoperative intestinal ileus. *Dis Colon Rect* 1961; 4:307-18.
12. Waldron GW and Hampton JM: Intestinal obstruction: A half century comparative analysis. *Ann Surg* 1961; 153:839-50.
13. McEntee G, Pender D, McCoullough M, et al: Current spectrum of intestinal obstruction. *Br J Surg* 1987; 74:976-80.
14. Lewis FR, Holcroft JW, Boey J, et al: Appendicitis: A critical review of diagnosis and treatment in 1,000 cases. *Arch Surg* 1975; 110:677-84.
15. Owens BJ and Hamit HF: Appendicitis in the elderly. *Ann Surg* 1978; 187:392-6.
16. Melody GF: Intestinal obstruction following gynecologic surgery. *Obstet and Gynecol* 1958;11: 139-147.
17. Sannella NA. Early and late obstruction of the small bowel after abdominoperineal resection. *Am J Surg* 1975; 130: 270-2.
18. Feinstein AR: *Clinical Epidemiology: The Architecture of Clinical Research.* Philadelphia: WB Saunders Company, 1985.
19. Kaplan EL and Meier P: Nonparametric estimation from incomplete observations. *J Am Stat Assoc* 1958; 53:457-81.
20. *International Classification of Diseases—Ninth Revision.* Washington, DC: United States Department of Health and Human Services, 1989.
21. Berk JE: *Gastroenterology.* Philadelphia: W. B. Saunders, Company, 1985.
22. Bradley EL and Isaacs J: Appendiceal abscess revisited. *Arch Surg* 1978; 113:130-2.
23. Dubois F, Icard P, Berthelot G, et al: Coelioscopic cholecystec–tomy: Preliminary report of 36 cases. *Ann Surg* 1990; 11:60-2.
24. Soper NJ, Barteau JA, Clayman RV, et al: Comparison of early postoperative results for laparoscopic versus standard open cholecystectomy. *Surg Gynecol Obstet* 1992; 174:114-8.
25. Soper NJ: Laparoscopic cholecystectomy: A promising new "branch" in the algorithm of gallstone management. *Surg* 1991; 109:342-4.

Because One Size Doesn't Fit All...

New Humulin 50/50 is the tailor-made answer to individual patient needs. A unique combination of equal amounts of Regular human insulin and NPH human insulin, it will be useful in situations in which a greater initial insulin response is desirable for greater glycemic control.

Like Humulin 70/30,* new Humulin 50/50 offers the convenience and accuracy of a premix. And it can be used in conjunction with an existing 70/30 regimen.



New Humulin® 50/50

50% human insulin isophane suspension
50% human insulin injection (recombinant DNA origin)

The Newest Option in Insulin Therapy

WARNING: Any change of insulin should be made cautiously and only under medical supervision.

*Humulin® 70/30 (70% human insulin isophane suspension, 30% human insulin injection [recombinant DNA origin]).

Global Excellence in Diabetes Care
Eli Lilly and Company
Indianapolis, Indiana
46285

HI-7911-B-249343 © 1992, ELI LILLY AND COMPANY

Postoperative Compartment Syndrome and the Lithotomy Position:
A Report of Three Cases and Analysis of Potential Risk Factors

J.P. MULHALL, M.D. AND A.D. DREZNER, M.D., PH.D.

ABSTRACT—There exists a definite association between the placement of patients in the modified lithotomy position and the development of post-operative compartment syndrome. It must be appreciated that this syndrome is potentially life-threatening and frequently results in long-term sequelae. We report three cases which illustrate the problem and analyze the multifactorial etiology of this condition and propose a strategy for its prophylaxis. The combination of direct pressure on the posterior compartment of the calf, elevation of the legs above heart level, a number of intraoperative variables, the preexistence of any lower limb arterial insufficiency, and in some cases, the use of intraoperative compression boots, may lead to a state of hypoperfusion in the anterior and posterior compartment musculature. Upon completion of the operation, removal from the lithotomy position, and correction of the etiological factors, a reperfusion occurs with the development of a capillary leak with subsequent tissue edema causing an increase in compartmental pressure which may result in neurovascular compromise. This is the compartment syndrome. Emphasis must be placed on the identification of high-risk patients, the prevention or rapid correction of any etiological factors, the early diagnosis of the problem, and an aggressive approach to its treatment.

Introduction

THE association between compartment syndrome and the positioning of patients during operations has long been appreciated. Indeed, the first such case was reported in 1953.[1] Thus far, a total of 15 cases have been reported, the majority secondary to positioning in the modified lithotomy position, but some the result of the knee-chest position. We report three cases of patients who were placed in the lithotomy position intraoperatively and who subsequently developed compartment syndrome, discuss the etiology of this complication, explore possible risk factors for its development, and suggest a strategy for prophylaxis.

Case Reports

1. A 66-year-old female was admitted to the hospital for a low anterior resection for carcinoma of the rectum. Her past medical history was unremarkable except for intermittent claudication in both limbs, right worse than left. The patient was brought to the operating room, and after the induction of general anesthesia was placed in the lithotomy position with stirrups. Compression boots were applied to both lower limbs prior to arrival in the operating room. The operation proceeded without incident and five hours later the patient was transferred to the recovery room. Within one hour of her arrival in the recovery room she was alert enough to complain of severe pain in her right lower leg, particularly the anterior aspect. Physical examination at that time revealed a tense anterior compartment, pain on dorsiflexion of the ankle, but normal sensation and pulses. She had emergency venous and arterial Doppler studies performed which revealed neither venous thrombosis nor arterial occlusion. By this time (two hours after arrival in the recovery room) she was complaining of hyposthesia and parasthesiae in the dorsum of her foot.

A clinical diagnosis of compartment syndrome was made and she was immediately returned to the operating room for emergency fasciotomies of her anterior and lateral compartments. The lateral compartment muscles appeared normal but the anterior compartment musculature was edematous although viable. The incisions were left open. Upon waking the patient reported complete

---

J.P. MULHALL, M.D., Department of Surgery and currently a resident in Urology, University of Connecticut School of Medicine, Farmington, and Department of Surgery, Section of Vascular Surgery, Hartford Hospital, Hartford. A.D. DREZNER, M.D., Ph.D., Department of Surgery, University of Connecticut School of Medicine, Farmington, and Department of Surgery, Section of Vascular Surgery, Hartford Hospital, Hartford.

Address for reprints: A.D. Drezner, M.D., 85 Seymour Street, Hartford, CT 06106.

relief of her previous pain. A urine test for myoglobin prior to the return to the operating room was positive and the patient was given intravenous fluids and mannitol until the urine was clear of myoglobin. Five days later the edema had resolved such that reapproximation of her fasciotomy wounds was possible. The patient was discharged on the 15th postoperative day; she was walking well without residual neurovascular deficits.

2. A 23-year-old white male with a long history of ulcerative colitis, including a history of a total abdominal colectomy with ileostomy, was admitted for takedown of his ileostomy with fashioning of an ileoanal pouch. For the operation he was placed in the lithotomy position using stirrups following the induction of general anesthesia. Compression boots had been applied to both lower limbs before the patient arrived in the operating room. The operation lasted six and a half hours and was without incident. The patient subsequently spent two hours in the recovery room before being removed to his room on the surgical floor. Three hours after the operation the patient complained of tenderness in his left calf. Two hours later, with an increase in pain, he was noted to have a swollen calf with marked tenderness on compression, although neurologically there were no abnormal findings and he had normal arterial pulses.

The patient was repeatedly examined and, after developing a mild sensory deficit in his left foot six hours later, he was returned to the operating room for decompression of his posterior compartment. A CPK level was elevated at 3,594 U/L. At fasciotomy the muscles of the posterior compartment were observed to be markedly edematous but viable. The fasciotomy wounds were left open. A urinalysis for myoglobin was positive and the patient was treated with intravenous fluids and mannitol. The following day the patient had some residual hyposthesia of the plantar surface of his left foot. The fasciotomy wounds were allowed to heal by secondary intention. The neurosensory deficit resolved over the course of the next week and the patient was discharged home on the ninth postoperative day without any neurovascular complaints.

3. A 66-year-old male was admitted for excision of a pelvic mass that had been discoved on routine physical examination. Following extensive radiologic imaging and biopsy, a diagnosis of a pelvic mesenchymal tumor was made. He was taken to the operating room for a pelvic exenteration with fashioning of an ileal conduit, sigmoid colostomy, and the placement of a pelvic Dexon mesh sling. The operation lasted eight hours and was performed entirely in the lithotomy position. The patient had compression boots applied preoperatively and wore them throughout the operation. The immediate postoperative course was unremarkable. The afternoon of the first postoperative day the patient began to complain of pain in his right calf. Physical examination at that time revealed tense anterior and posterior muscle compartments with diminished sensation in his whole foot, maximally in the first interdigital web space. A venous Doppler ultrasound was negative for venous thrombosis.

The patient was returned to the operating room for the performance of fasciotomies of all four compartments. On making the incisions the muscles were noted to be edematous but viable. The fasciotomy wounds were left open. A CPK level prior to his return to the operating room was 13,000 U/L, and the patient's urine was positive for myoglobin. He was aggressively rehydrated and was given mannitol until his urine was clear of myoglobin five days later. Over the course of the next three days the CPK level returned to normal and by the end of the first postoperative week the residual hypesthesia in his foot had completely resolved. Eleven days postoperatively the patient returned to the operating room for the placement of a split-thickness skin graft to the lateral fasciotomy wound. He recovered from all aspects of the procedures and was discharged home three weeks postoperatively. On follow-up he has no sequelae from his compartment syndrome.

Discussion

Since 1953 only 15 cases of postoperative compartment syndrome have been reported in the literature. Ten of these have been in cases using the lithotomy position and four in cases using the knee-chest ("tuck") position. The cases placed in the lithotomy position were a combination of general surgical,[2] colorectal,[3] urologic,[4-9] gynecologic,[10] and orthopedic[11] operations, while all the cases utilizing the "tuck" position were laminectomies performed by a neurosurgical service.[1,12,13] All of these cases lasted longer than six hours and all except three had bilateral lower limb involvement. Two of the cases with unilateral compartment syndrome were in patients placed in the hemilithotomy position for the internal fixation of femoral fractures. All cases had some form of residual deficit on discharge from hospital, ranging from renal failure to neurological deficits to skin and muscle necrosis. Indeed, the first reported case was of a patient placed in the "tuck" position who later developed the renal crush syndrome with renal failure secondary to myoglobinuria and subsequently died.[1]

Postoperative compartment syndrome is a reperfusion injury. A transient reduction in the perfusion pressure to the tissues of a compartment is followed by a period of reopening of that blood supply resulting in cellular swelling with capillary leakage and subsequent massive tissue edema. This edema may result in venous occlusion with perpetuation of the cycle, causing neural compromise, and if allowed to continue, eventual arterial occlusion.

It has been shown that at rest the pressure within the calf compartments is normally below 20 mm Hg.[14] There is debate concerning the pressure required to cause a reduction in perfusion pressure sufficient to result in a compartment syndrome. Pressures as high as 50-55 mm Hg for four to eight hours have been cited to result uniformly in compartment syndrome[14] while other workers have claimed that 35-40 mm Hg for a similar durations is suffiecient.[13] Intraoperative pressures as high as 240 mm Hg have been recorded in patients placed in the lithotomy position.[14] Furthermore, the pressure required is inversely related to the duration spent at this pressure. Even more importantly the patient's baseline perfusion pressure must be taken into account. For example, a patient with a perfusion pressure of 100 mm Hg at the calf level will require greater occlusion pressures and a longer exposure to these than a patient with peripheral vascular disease with calf pressures of 40 mm Hg.

The modified lithotomy position is obviously apt to cause compartment syndrome because of the pressure exerted on the calf muscles by some support stirrups, resulting in a decrease in the size of the compartment. It is important to remember other factors that may also play a role in the development of this syndrome, such as episodes of intraoperative hypotension, fluid deficit that may result in a low-flow state, the presence of acidemia, or the administration of drugs with vasoconstrictive effects. All of these factors are also at play in the development of postoperative compartment syndrome in patients who have been placed in the knee-chest position.

Thus far all reported cases following the lithotomy position were of at least six and one half hours duration, and three hours duration for the knee-chest position. Two of our cases were over six hours long, but one was only five hours in duration. The latter case is of interest in that the development of the syndrome was unilateral, occurring only in the patient's right lower limb. Of note, this patient had preexisting peripheral vascular disease with preoperative ankle-brachial indices of 0.27 in her right leg and 0.34 in her left leg. Undoubtedly, placing this patient in the lithotomy position put her at greater risk of developing the compartment syndrome, particularly in her right leg. This suggests the importance of avoiding placing such patients in the lithotomy position for any operation irrespective of the nature or duration. Furthermore, the use of compression boots in these patients we feel is contraindicated.

Clearly of great importance is the nature of the stirrups that are used to support the legs during lithotomy positioning. While the elevation of the legs above heart level is unavoidable the direct pressure on the calf muscles by the stirrup cushions is certainly preventable by using stirrups of the Allen type where the feet are supported but no pressure is exerted on the muscles of the posterior compartment. Furthermore, it is essential to avoid any circumferential wrapping of the legs which would only further compromise arterial inflow. In all of our cases calf-supporting stirrups were used and although extensive calf padding was used this still remains a poor substitute for the use of foot-supporting stirrups.

In all three of our patients compression boots were applied preoperatively, and were worn throughout the operation. The compression appliances used were of the intermittent sequential pressure variety (Kendall, Mansfield, Mass.). This system allows the application of pressure via bags in sequence over the calf muscles. The compression time is 12 seconds each minute with a rest phase of zero pressure for the remainder of the minute. The pressure attained during compression is adjustable in a range between 35-50 mm Hg and can be controlled by the operating room personnel.

Malfunction of such a system, as has already been reported in the literature[8] would obviously have catastrophic effects, but even under normal circumstances the system may cause problems in patients with already low perfusion pressures such as in our first patient with peripheral vascular disease. Even a compression phase of as little as 12 seconds each minute with a pressure of 35 mm Hg in a patient with an ankle-brachial index of 0.3 may generate enough ischemia to result in a compartment syndrome. Exactly what role compression boots have in the genesis of postoperative compartment syndrome in the normal patient has not been defined, and will require further evaluation.

At this time, there is no way to monitor these patients intraoperatively without using a continuous intracompartmental transducer. This is an invasive procedure with a low but existing risk and requires additional personnel for the operation of the equipment. Furthermore, if we were to subject every patient who we felt was at risk for the development of postoperative compartment syndrome to this surveillance, it would prove very costly. A simpler means of monitoring such patients may be the use of simple pulse oximetry of the toes. In theory, a desaturation in such a system should alert us to a perfusion problem, all other factors remaining stable. This would give us a headstart in the identification of high-risk patients for postoperative surveillance, and in some cases allow us to correct the problem in the operating room.

In this report none of our patients suffered any long-term disability after of discharge from hospital, unlike those cases reported in the literature thus far. Sequelae to this complication range in severity from the devastating renal crush syndrome with rhabdomyolysis of sufficient magnitude to cause gross myoglobinuria and subsequent renal failure, to the less serious but nevertheless signifi-

HEALTH SCIENCES LIBRARY

cant neurological effects such as hyposthesia or weakness of ankle plantar and/or dorsiflexion. Other problems such as skin or muscle necrosis and fibrosis have also been reported, which themselves present a major long-term rehabilitation problem. It is noteworthy that none of our patients had any such problems. One reason for this was the aggressive approach taken to the performance of fasciotomies in our patients. In one patient the problem was diagnosed within three hours, in a second within six hours of completion of the operation, while the third was diagnosed on the afternoon of the first postoperative day. All of our patients returned to the operating room for fasciotomies within an hour of diagnosis.

The key to the diagnosis of compartment syndrome is a high level of suspicion. The diagnosis is a purely clinical one. Once any combination of the classical symptoms is present, such as calf or anterior muscle compartment pain, calf or pedal edema, exacerbation of pain on passive movement of the affected muscle groups, and neurological symptoms such as pedal hyposthesia, an urgent return to the operating room is justified and indeed mandatory. It is important to appreciate that the development of motor nerve symptoms or signs and the loss of arterial pulses are very late signs that indicate that irreparable damage has already occurred.

All of these patients should have their urine checked for myoglobin, and, if present, appropriate steps should be taken to hydrate the patient fully and consideration should be given to the use of mannitol and/or the alkalinization of the urine. While mannitol causes a forced osmotic diuresis its main function in this syndrome, as with any other reperfusion injury, lies in its utility as a superoxide scavenger. Many advocate the evaluation of serum CPK levels. We feel they neither aid the diagnosis nor influence the decision concerning the performance of fasciotomies. Deep venous thrombosis and arterial occlusion are frequently listed as differential diagnoses of the postoperative compartment syndome, but in a patient immediately postoperative, especially one in whom nonphysiological positioning has been used, a diagnosis of compartment syndrome must be entertained and ruled out. We feel therefore that venous or arterial Doppler ultrasound studies are purely complementary to clinical examination and in no way replace clinical evaluation of the patient.

Some debate exists over the role of compartmental pressure measurement in the evaluation of the patient with early postoperative lower leg pain. Our feeling is that in situations where the classical combination of symptoms and signs exist there is little role for such a study as one must surely already be committed to performing fasciotomies and must continue with this plan irrespective of the absolute intracompartmental pressure recorded. Where postoperative compartmental pressure measurement may be of great benefit is in cases in which there is doubt about the diagnosis, and pressures that are elevated above normal may persuade the clinician to proceed with fasciotomies. This may be especially true where the patient is still somnolent or even paralysed, has a swollen tense anterior or posterior compartment, but cannot or does not complain of any of the classical symptoms. In retrospect the patient in our second case would probably have benefited from the early measurement of intracompartmental pressures prior to the development a neurosensory deficit. It is important to appreciate that the existence of any neurosensory or motor deficit may herald the presence of already irreversible damage to the musculature and neural components of that compartment. Finally, the determination of preoperative ankle-brachial indices combined with pedal pulse oximetry in patients who require an operation in the lithotomy position may help identify a subgroup at high risk.

Summary

We have reported three cases of a syndrome that is probably more common than many realize. It is impossible to estimate how many patients have mild, subclinical, unrecognized forms of the syndrome. We emphasize the need to select carefully those patients who are especially at risk from the use of the lithotomy position and the use of compression boots. We point out that a number of intraoperative factors may play larger roles than previously realized. It is important to be aware of this condition and take appropriate measures to prevent or treat it in a timely fashion. We suggest that the use of preoperative ankle-brachial index evaluation and pedal pulse oximetry may be helpful in the identification of at-risk patients. Finally, we reiterate that the key step in the management of this problem is awareness and early aggressive treatment.

REFERENCES

1. Gordon BS, Newman W: Lower nephron syndrome following prolonged knee-chest position. *J Bone Joint Surg* 1953; 35A: 764-7.
2. Lydon JC, Spielman FJ: Bilateral compartment syndrome following prolonged surgery in the lithotomy position. *Anesthesiology* 1984; 60:236-8.
3. Khalil IM: Bilateral compartmental syndrome after prolonged surgery in the lithotomy position. *J Vasc Surg* 1987; 5:879-81.
4. Bergqvist D, Bohe M, Ekelund G, et al: Compartment syndrome after prolonged surgery with leg supports. *J Colorect Dis* 1990; 5:1-5.
5. Hellblom B, Scharnagl E, et al: Kompartmentsyndrom als Lagerungskomplikation bei Penisplantation. *Handchirurgie* 1986; 18:363-6.
6. Leff R, Shapiro SR: Lower extremity complications of the lithotomy position: Prevention and management. *J Urol* 1979; 122;138-9.

7. Reddy PK, Kaye KW: Deep posterior compartmental syndrome: A serious complication of the lithotomy position. *J Urol* 1984; 132:144-5.
8. Werbel GB, Shybut GT: Acute compartment syndrome caused by a malfunctioning pneumatic-compression boot. *J Bone Joint Surg* 1986; 68A:1445-6.
9. Goldberg M, Stecker J, Scarff JE, et al: Rhabdomyolysis associated with urethral stricture repair: Report of a case. *J Urol* 1980; 124:730-1.
10. Adler MA, Loughlin JS, Morin CJ, et al: Bilateral compartment syndrome after a long gynecologic operation in the lithotomy position. *Am J Obstet Gynecol* 1990; 162:1271-2.
11. Dugdale TW, Schuker S, Deafenbough MK, et al: Compartment syndrome complicating use of the hemi-lithotomy position during femoral nailing. *J Bone Joint Surg* 1989; 71-A:1556-7.
12. Keim HA, Weinstein JD: Acute renal failure—a complication of spinal fusion in the tuck position. *J Bone Joint Surg* 1970; 52A:1248-50.
13. Aschoff A, Steiner-Milz H, Steiner HH: Lower limb compartment syndrome following lumbar discectomy in the knee-chest position. *Neurosurg* Rev. 1990; 13:1559.
14. Mubarak SJ, Hargens AR: Acute compartment syndromes. *Surg Clin N Am* 1983; 3:539-65.

Lyme Disease

The Lyme Care Center seeks qualified physicians throughout Connecticut to whom patients presenting symptoms of early, middle stage and chronic Lyme disease can be referred for treatment. Candidates for referrals must treat aggressively, appropriately, and compassionately. To those physicians who qualify for inclusion in our network, we offer a tremendous opportunity for practice enhancement. **Interested physicians should contact Joseph A. Hernandez, Managing Director.**

1-800-842-5248

HoMed/The Lyme Care Center
2371 Boston Post Road
Guilford, CT. 06437-2315

HoMed

THE LYME CARE CENTER SM

CALL FOR PAPERS

Members of the Connecticut State Medical Society reading papers before other organizations are requested to submit their papers to *Connecticut Medicine* for consideration by the Board of Editors for publication. Authors preparing manuscripts for submission to the Journal should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office. Adherence to the instructions will prevent delays both in acceptance and in publication.

Note: Manuscripts set on almost any computers word processer (eg, Wordperfect) can be submitted on disk (5-1/4" or 3-1/2") along with a hardcopy of the manuscript.

Please send to:

Robert U. Massey, M.D.
Connecticut Medicine
160 St. Ronan Street
New Haven, CT 06511

For certain categories of taxpayers, we are still accepting contributions that will be deductible for 1992.

ENDORSED BY THE MEDICAL SOCIETY OF NEW JERSEY

**V.E.B.A. PLAN*

FEATURES

TAX DEDUCTIBLE CONTRIBUTIONS TO AN I.R.S. APPROVED TRUST

EVEN IF ...

- **YOUR PENSION PLAN IS OVER FUNDED**
- **YOUR PENSION PLAN IS MAXIMUM FUNDED**
- **YOU HAVE NO PENSION PLAN**

PLEASE SEND ME A copy of your informational pamphlet on the * V.E.B.A. program. I understand there is no obligation.

NAME______________________________

ADDRESS______________________________

DATE OF BIRTH________________ TELEPHONE # (____)________________

Mail to:

THE KIRWAN COMPANIES
402 MIDDLETOWN BLVD., SUITE 202
LANGHORNE, PA. 19047

(800) 283-7666........................ (215) 750-7616........................ FAX (215) 750-7791

*VOLUNTARY EMPLOYEE BENEFICIARIES ASSOCIATION

Pregnancy Related Coronary Artery Dissection: A Case Report and Collective Review

DANIEL T. ENGELMAN, M.D., JOHN THAYER, M.D., JOHN DEROSSI, M.D.,
JACOB SCHEINERMAN, M.D., AND LOUIS BROWN, M.D.

ABSTRACT—There have been 31 patients with pregnancy related coronary artery dissection reported in the medical literature. We report a case of both right and left coronary artery dissection in a postpartum patient. A collective review of the clinical course, risk factors, and treatment specific mortality rates of coronary artery dissection in near term and postpartum females is discussed. The mean age was 33, the mean parity was 2.7, and the mean time span was 20 days postpartum. Twenty-two percent had known risk factors for coronary artery disease. Eighty-four percent involved the left anterior descending artery. Overall mortality was 66%. Of the patients that were alive on admission, 61% were treated medically (n=11) with a 64% mortality; 39% were treated surgically (n=7) with a 0% mortality. We conclude that pregnancy related coronary artery dissection occurs in otherwise healthy women near term or within three months postpartum. Multiparity and advanced maternal age may be risk factors. Early diagnosis and prompt surgical treatment may improve outcome.

Introduction

SPONTANEOUS coronary ártery dissections are a rare cause of ischemic heart disease and sudden death. In a recent review, 31% of all primary coronary dissections occurred in patients who were near term or in the puerperium. While spontaneous coronary artery dissections are known to occur in association with Marfan's syndrome, angiitis, trauma, sarcoidosis, coronary angiography, percutaneous transluminal angioplasty, and surgery, most are idiopathic.[1]

The first case of pregnancy related coronary artery dissection in the medical literature was reported by Lovitt in 1952. Since that time there have been a total of 31 patients reported with this disease. The majority of patients died as a direct result of the dissection. A wide range of treatment modalities have been used, with varying results. This diagnosis must be considered early in postpartum women with symptoms of myocardial ischemia, as prompt treatment may be lifesaving. In this paper we present a case of coronary artery dissection occurring five days postpartum and review the collective literature. To our knowledge this is only the second reported case of spontaneous right and left coronary artery dissection and the first in which the patient survived.

Case Report

A 32-year-old, gravida 3, para 2 woman had an uncomplicated caesarean section for macrosomia. Five days following her surgery she developed an acute onset of substernal chest pain with radiation to her neck and left arm. Physical examination revealed a blood pressure of 120/60, pulse rate of 55/minute, and a respiratory rate of 20. An EKG demonstrated a sinus bradycardia, but was otherwise normal. The patient's pain quickly dissipated and she was discharged home that afternoon.

During the next three days the patient did not experience any chest pain. On postoperative day eight, the patient had a severe episode of chest pain with radiation to both arms, nausea, vomiting, shortness of breath, and diaphoresis necessitating an evaluation in the emergency room. She had no significant past medical history. The patient's only known risk factor for cardiovascular disease was a positive family history. There was no history

---

DANIEL T. ENGELMAN, M.D., Resident, General Surgery, University of Connecticut School of Medicine. JOHN THAYER, M.D., Assistant Clinical Professor of Medicine, University of Connecticut School of Medicine. JOHN DEROSSI, M.D., Assistant Clinical Professor of Medicine, University of Connecticut School of Medicine. JACOB SCHEINERMAN, M.D., Assistant Clinical Professor of Medicine, University of Connecticut School of Medicine. LOUIS BROWN, M.D., Assistant Clinical Professor of Medicine, University of Connecticut School of Medicine.

From the Department of Surgery, University of Connecticut School of Medicine, 263 Farmington Avenue, Farmington, Connecticut, 06030-3955.

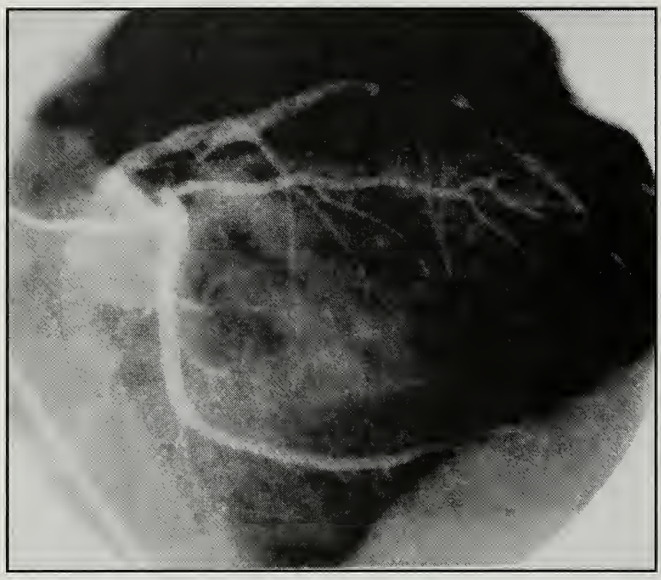


Figure 1
RAO projection following injection of left main coronary artery demonstrating dissection in the proximal LAD.

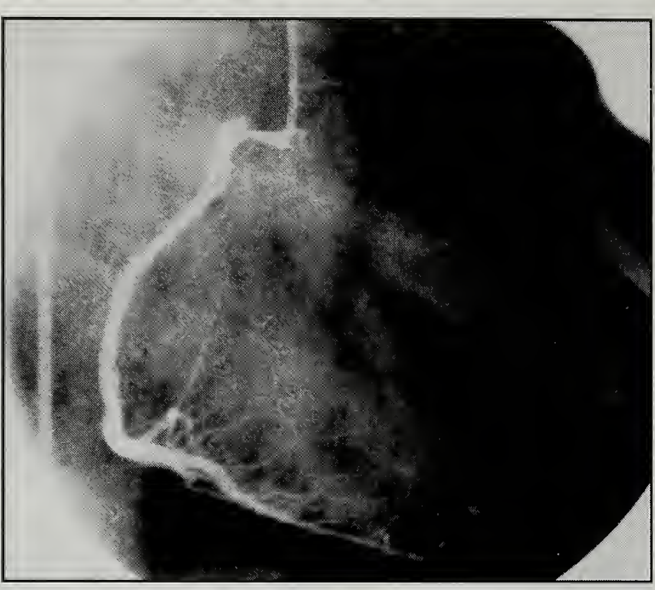


Figure 2
RAO projection of right coronary artery demonstrating spiral dissection of proximal and mid RCA.

of Kowasaki's disease or hyperextensible joints. Physical examination at that time revealed a blood pressure of 140/80, pulse rate of 65/minute, a respiratory rate of 14, and a normal cardiovascular exam. A 12-lead EKG was remarkable for 2-3mm ST depression in leads V3 through V6, mild ST elevation in leads II, III, and aVT, T wave inversion in leads I and aVL, and PVCs in bigeminy. The chest film was unremarkable. The possibility of a pulmonary embolus was entertained, but a V/Q scan was unremarkable. A myocardial infarction was confirmed by serial creatinine phosphokinase isoenzymes with a peak of 1438 with an 8% MB fraction. An echocardiogram revealed some posterior wall hypokinesis but was otherwise unremarkable. Over the next few days the patient continued to have recurrent chest pain despite treatment with nifedipine, Lopressor,® nitroglycerine, and heparin. On the morning of her fifth hospital day she awoke diaphoretic, complaining of crushing chest pain radiating down both of her arms. An EKG was obtained which demonstrated a sinus rhythm at a rate of 62 with ST depression in leads I, aVL, V5, and V6, ST elevation in lead III, and T wave inversion in lead I. She then underwent coronary angiography.

The catheterization revealed a dissection in the left anterior descending artery beginning prior to the first septal perforator. (See Fig. 1) The right coronary artery also had a dissection beginning just distal to the ostium and extending in a spiral fashion down two-thirds of this vessel with preservation of the distal flow. (See Fig. 2) The left main and circumflex coronary arteries were normal. A left ventricular angiogram disclosed marked hypokinesis of the posterior lateral wall with preserved septal motion. The patient was then taken emergently to the operating room.

Intraoperatively the patient had two saphenous vein grafts placed in the usual fashion without ligation of the dissecting coronary arteries; one to the left anterior descending (LAD) and one to the right coronary artery (RCA). The operation was complicated by ventricular fibrillation upon opening the chest requiring internal cardiac massage and the rapid initiation of cardiopulmonary bypass. The patient's heart continued to fibrillate until placed on cardiopulmonary bypass. Because of marked hemodynamic instability, saphenous vein bypass grafts were used as a conduit although internal mammary arteries would have been preferred. Aortic specimens did not show any histologic evidence of cystic medial degeneration or angiitis. The patient had an uncomplicated postoperative course and was discharged to home on postoperative day seven. A follow-up echocardiogram was obtained 19 days postoperatively. It demonstrated left ventricular dilation, abnormal septal wall motion, a minimal pericardial effusion, no valvular abnormalities, and a low normal ejection fraction.

Discussion

Including this case, there are 32 cases of pregnancy related spontaneous coronary artery dissections reported in the medical literature. The cases are summarized in Table I. Among the patients in whom it was reported, the average age was 33 and the average parity was 2.7. Two of the patients were near term, the rest were within three months of delivery. The average patient was 20 days postpartum. Only 22% (7/32) of the patients had known risk factors for heart disease.

Sixty-nine percent (22/32) of the patients were reported to have complained of chest pain as their first symptom. In 84% (27/32) of the cases, the LAD was involved in the dissection. To the authors' knowledge, this is only the second reported case of both right and left coronary artery dissection in the postpartum period, and the first in which the patient survived. A myocardial infarction was seen in 70% (19/27) of the cases that mentioned this entity.

The mortality of this disease was 66% (21/32). In 44% (14/32) of the cases the patient was dead on arrival to the hospital. In all but two of these cases, the patient's death occurred within two weeks of their first symptoms. Of those patients that were alive on admission to the hospital, a premorbid diagnosis of coronary dissection was made only 56% (10/18) of the time. In one of these cases the diagnosis was only made after the patient had her second infarction. It is interesting to note that six of the last seven cases reported in the literature were diagnosed prior to autopsy. The one missed diagnosis was made by the pathologist after the patient was successfully treated with a heart transplant.

Medical treatment was used in 61% (11/18) of the patients. Surgical treatment was used in the remaining 39% (7/18), including two patients that were initially managed medically. Of the surgically treated patients, four had coronary artery bypass grafts, two underwent heart transplants, and one had a surgical clot extrusion.

Histopathology

Coronary dissections typically begin within 2 cm of the aortic ostium and are limited to a 2 to 4 cm segment. Occasionally the dissections were noted to extend to the more distal parts of the vessel. Histologically, the dissection usually begins within the outer portion of the media. As the media fills with blood, the lumen is compressed thereby inhibiting distal coronary flow. The local adventitia is often secondarily infiltrated with neutrophils, eosinophils, and histiocytes.[2]

Pathogenesis

The definitive etiology of postpartum coronary dissections has not been determined. No other risk factors such as hypertension, atherosclerotic disease, family history, or abnormal connective tissue production have been elucidated. Advancing maternal age and multiparity appear to increase the risk.[3] This review supports that hypothesis.

Straining and the sheering forces encountered during labor and delivery may initiate a "two-stage process" causing an initial intimal rupture with subsequent hemorrhage into the media of the coronary vessels at a later stage. It has been suggested that clotting changes that occur early in the puerperium may delay medial bleeding initiated at delivery.[4] This "two-stage process" was found in three of the patients.[5] However, most patients have not been found to have intimal tears on pathological examination.

It has been shown that during pregnancy arterial walls undergo changes that may predispose to dissection. The changes include fragmentation of reticulum fibers, hypertrophy of smooth muscle cells, and loosening of the ground substance.[5] Changes in the media of coronary arteries of pregnant women have also been implicated.[5] Heefner reported a case of spontaneous coronary artery dissection in which the use of oral contraceptives was implicated.[6] High levels of circulating estrogens may be responsible for an increase in acid mucopolysaccharides in all connective tissues.[6] Bonnett has reported a case of postpartum dissection in which the collagen production in this patient was found to be decreased in an in vitro culture. It was thought that the low collagen synthesis might be responsible for an alteration in coronary vessel walls, thereby leading to spontaneous coronary artery dissection. The alteration in the collagen metabolism could be due to an altered endocrine status during pregnancy. These changes would be expected to return to normal during the three months following delivery.[7] Finally, a relative copper deficiency, which has been shown to cause vascular wall connective tissue defects in animals, has been proposed as a possible causative factor.[5]

Management

Various treatment options have been attempted in patients with postpartum coronary artery dissections. The first 12 cases were diagnosed at autopsy and either died suddenly or following medical management. In 1970 the first patient to have a premorbid diagnosis of a postpartum coronary artery dissection underwent a successful left ventricular aneurysmectomy and aortoposterolateral circumflex branch saphenous vein graft.[8]

Most of the literature on the management of acute coronary dissections comes from those that were angiographically induced. Surgical intervention is indicated in those cases which demonstrate evolving dissections and luminal narrowing resulting in significant hemodynamic compromise. This usually requires placement of a coronary artery bypass graft using the saphenous vein or an internal mammary artery. However, there are two reported cases in which patients were treated successfully with heart transplants.[4,9]

Women who present in the puerperium with symptoms of ischemic heart disease represent a special class of cardiac patient. The diagnosis of spontaneous coronary artery dissection must be entertained early in the clinical course. Although there are only 31 published cases, there is evidence to suggest that the incidence may be higher

Table 1

Postpartum Coronary Artery Dissections

Pt. # (ref)	*Year*	*Age*	*Mat Par*	*Date of Symptoms*	*Risk Factors*	*First Symptoms*	*Arteries involved*	*MI*	*DX*	*Treatment*	*Outcome (Time from sx)*
Medical Treatment											
1(5)	1952	39		2 wk pp		CP,Dyspnea	LAD		-	Medical	Death(2.5 hrs)
2(11)	1960	42		6 wk pp	HTN	CP,Nausea	LAD	-	-	Medical	Death(1 yr)
3(5)	1962	35		6 wk pp			LAD			DOA	S.D.
4(5)	1965	40		2.5 wk pp		CP	LAD	+		DOA	S.D.
5(5)	1965	36		19 d pp		Collapse	LAD			DOA	S.D.
6(5)	1965	41		80 d pp		Collapse	LAD			DOA	S.D.
7(12)	1969	31	9	16 d pp		CP	LM,LAD,LCX	-		DOA	Death(10 hrs)
8(5)	1970	39	3	9th month		CP,Nausea	LM,LAD,LCX	+	-	Medical	Death(2.5 hrs)
9(5)	1970	39	3	9th month		None	LAD	-		DOA	S.D.
10(5)	1970	26	4	2 wk pp		CP	LAD	-	-	Medical	Death(4 hrs)
11(13)	1971	27	3	2 wk pp		Collapse	LAD	-		DOA	S.D.
12(14)	1972	32		6 d pp		CP	LAD	+		DOA	Death(14 days)
13(15)	1978	40	4	5 wk pp	HTN	CP	RCA			DOA	Death(10 days)
14(15)	1978	35	1	8 wk pp	Rheu. fever	CP	LCA	+	-	Medical	Death(30 hrs)
15(16)	1978	28	2	3 wk pp	HTN,tobac.	CP	LAD,LCX	+	+	Medical	Alive(4 mos)
16(17)	1979	37	5	4 wk pp		CP	RCA	+		DOA	S.D.
17(18)	1980	27	2	16 d pp		CP	RCA,LAD	-		DOA	Death(24 hrs)
18(19)	1982	26		4 wk pp			LAD			DOA	S.D.
19(20)	1984	27	4	0 d pp		CP	LM,LAD,LCX	+	-	Medical	Death(5 days)
20(21)	1986	37		8 d pp		CP	LCX	+		DOA	S.D.
21(22)	1987	27		3 m pp		Collapse	RCA	+		DOA	S.D.
22(23)	1988	46		0 d pp			RCA	+	+	Medical	Alive(36 mos)
23(24)	1988	28	2	1 d pp	HTN,chol.	CP	LAD	+	+	Medical	Death(48 mos)
24(25)	1989	39		4 d pp			LAD	+		Medical	Alive(5 mos)
25(26)	1989	32	0	2 d pp	plac. previa	CP	LAD	+	+	Medical	Alive
Operative Treatment											
26(8)	1975	31		3 d pp		CP	LM,LAD LCX	+	+	Med/CABG	Alive(4 yrs)
27(27)	1982	33	1	12 d pp		CP	LM,LAD	+	+	CABG	Alive
28(7)	1986	32	1	12 d pp		CP	LAD,LCX	+	-/+	Med/CABG	Alive(18 mos)
29(28)	1986	33	2	0 d pp	tobac.	CP	LM,LAD,LCX	+	-	Surg. *	Alive(15 mos)
30(9)	1989	29		3 wk pp		CP	LAD,LCX	+	+	Transpl	Alive
31(4)	1990	33	1	4 d pp		Epigastric pain	LM,LAD	+	-	Transpl	Alive(36 mos)
32(†)	1991	32	2	5 d pp	fam hist.	CP	RCA,LAD	+	+	CABG	Alive(12 mos)

Key:
chol = hypercholesterolemia
DX = Was a premorbid diagnosis made?
LCX = left circumflex
MI = myocardial infarction
S.D. = sudden death
tobac = tobacco smoker
CP = chest pain
HTN = hypertension
LM = left main
pp = postpartum
Surg * = This patient was treated with a surgical clot extrusion
† = present case
DOA = dead on arrival to hospital
LAD = left anterior descending
Mat Par = maternal parity
RCA = right coronary artery

than reported. There are three reports of myocardial infarctions during pregnancy without subsequent evidence of vessel obstruction at angiography. This suggests that the dissections may have healed by the time the catheterizations were performed.[9]

The initial management of these patients who present with symptoms of ischemic chest pain and associated laboratory or EKG abnormalities is with heparin and nitroglycerin. The nitroglycerin acts to abate any associated spasm and the heparin helps to prevent thrombosis of an already narrowed vessel. More aggressive medical modalities such as thrombolytics and percutaneous transluminal coronary angioplasty are probably contraindicated in this patient population at high risk for a primary dissection. Early angiography in this select group is indicated as myocardial salvage may depend on urgent bypass surgery. Surgical intervention may be guided by generally accepted criteria. If there is significant luminal narrowing of a coronary vessel supplying a large amount of territory as well as objective evidence of ischemia, then surgery is indicated. About 80% of the dissections involve the proximal LAD while the remainder involve the proximal RCA.

Our case is very unusual as it involved dissections of both the LAD and RCA. It is interesting to speculate that her original chest pain may have represented a dissection of one of her arteries and that the subsequent hemodynamic collapse was caused by the second dissection.

There is some discussion in the literature regarding the advisability of ligating the dissected coronary vessel. The general consensus appears to be that proximal ligation is not required. This approach would make the distal vessel totally dependent on the bypass graft.[1] Additionally, there are reports of healing of the dissections with restored patency of the native vessel.[10]

In the case of a distal dissection, the layers of the vessel should be carefully approximated during anastomosis. Tacking sutures may be placed posteriorly to secure the vessel layers if the dissection involves the posterior wall. The anastomosis should otherwise be placed at the technically most feasible point. Because most of the patients with primary coronary dissection are young, internal mammary grafts are favored because of their higher patency rates.

Conclusion

Two presentations of this disease are generally seen. Either the patient has a sudden death and the diagnosis is made at autopsy, or the patient survives a myocardial infarction allowing the diagnosis to be made at angiography. In many cases, the initial hemorrhage appears to take place some time before the final calamity. Therefore, we recommend early coronary angiography in women who develop angina in the puerperium. This will prevent delayed diagnoses and facilitate the differentiation between subsequent medical *vs* surgical treatment of this disease. This is especially true of those patients with proven acute myocardial infarctions, as current treatment regimens, such as fibrinolytics, may be contraindicated. It should be noted however, that early angiography has on occasion resulted in a new dissection with subsequent reinfarction.[10]

REFERENCES

1. Thayer JO, Healy RW, Maggs PR: Spontaneous coronary artery dissection. *Ann Thorac Surg* 1987; 44:97-102.
2. Smith JC: Dissecting aneurysms of coronary arteries. *Arch Pathol* 1975; 99:117-21.
3. Barrett JM: Pregnancy-related rupture of arterial aneurysms. *Obstet Gynecol Surv* 1982; 37:557-66.
4. Curiel P, Spinelli G, Petrella A, et al: Postpartum coronary artery dissection followed by heart transplantation. *Am J Obstet Gynecol* 1990; 163:538-9.
5. Asuncion CM, Hyun J: Dissecting intramural hematoma of the coronary artery in pregnancy and the puerperium. *Obstet Gynecol* 1972; 40:202-10.
6. Heefner WA: Dissecting hematoma of the coronary artery. *JAMA* 1973; 223:550-1.
7. Bonnet J, Aumailley M, Thomas D, et al: Spontaneous coronary artery dissection: Case report and evidence for a defect in collagen metabolism. *Eur Heart J* 1986; 7:904-9.
8. Razavi M: Unusual forms of coronary artery disease. *Cardiovasc Clin* 1975; 7:25-46.
9. Movsesian MA, Wray RB: Postpartum myocardial infarction. *Br Heart J* 1989; 62:154-6.
10. Connors JP, Thanavaro S, Shaw RC, et al: Urgent myocardial revascularization for dissection of the left main coronary artery. *J Thorac Cardiovasc Surg* 1982; 84:349-52.
11. Wells AL: Dissecting aneurysm of coronary artery in the puerperium. *J Path Bact* 1960; 79:404-5.
12. Palomino SJ: Dissecting intramural hematoma of left coronary artery in the puerperium. *Am J Clin Pathol* 1969; 51:119-25.
13. DiMaio VJ, Di Maio DJ: Postpartum dissecting coronary aneurysm. *NY State J Med* 1971; 71:767-9.
14. Claudon DG, Claudon DB, Edwards JE: Primary dissecting aneurysm of coronary artery. *Circulation* 1972; 45:259-66.
15. Jewett JF: Two dissecting coronary-artery aneurysms post partum. *N Engl J Med* 1978; 298:1255-6.
16. Shaver PJ, Carrig TF, Baker WP: Postpartum coronary artery dissection. *Br Heart J* 1978; 40:83-6.
17. Pisano RV, Taylor MB, Sopher IM: Dissecting coronary artery aneurysm: A report of two cases. *J Forensic Sci* 1979; 24:18-25.
18. Ehya H, Weitzner S: Postpartum dissecting aneurysm of coronary arteries in a patient with sarcoidosis. *South Med J* 1980; 73:87-8.
19. Robinowitz M, Virmani R, McAllister HA: Spontaneous coronary artery dissection and eosinophilic inflammation: A cause and effect relationship? *Am J Med* 1982; 72:923-8.
20. Johnson KM, Carpenter PL, Gaeckle T: Twenty-seven-year-old postpartum gravida IV para IV with hypotension and chest pain. *S D J Med* 1984; 37:17-22.
21. Sage MD, Koelmeyer TD, Smeeton WMI: Fatal postpartum coronary artery dissection. *Am J Forensic Med Pathol* 1986; 7:107-11.
22. Dowling GP, Buja M: Spontaneous coronary artery dissection occurs with and without periadventitial inflammation. *Arch Pathol Lab Med* 1987; 111:470-2.
23. Nishikawa H, Nakanishi S, Nishiyama S, et al: Primary coronary artery dissection observed at coronary angiography. *Am J Cardiol* 1988; 61:645-8.
24. Nishikawa H, Nakanishi S, Nishiyama S, et al: Primary coronary artery dissection: Its incidence, mode of onset and prognostic evaluation. *J Cardiol* 1988; 18:307-17.
25. DeMaio SJ, Kinsella SH, Silverman ME: Clinical course and long-term prognosis of spontaneous coronary artery dissection. *Am J Cardiol* 1989; 64:471-4.
26. Lette J, Gagnon A, Cerino M, et al: Apical hypertrophic cardiomyopathy with spontaneous postpartum coronary artery dissects. *Can J Cardiol* 1989; 5:311-4.
27. Chokron R, Thomas D, Leprince JL, et al: Dissection primitives isoleés des artères coronaires. À-propos de 3 cas d'évolution favorable. *Arch Mal Coeur* 1983; 76:886-92.
28. Vicari R, Eybel C, Monson D: Survival following spontaneous coronary artery dissection: Surgical repair by extrusion of intramural hematoma. *Am Heart J* 1986; 111:593-4.

Abstracts: 30th Annual Orthopaedic Conference

NEWINGTON CHILDREN'S HOSPITAL

JOHN V. BANTA, M.D.
Director of Orthopaedic Surgery, Newington Children's Hospital; Professor,
Department of Orthopaedic Surgery, University of Connecticut School of Medicine

FOREWORD—on 23 October 1992, the 30th consecutive Annual Orthopaedic Conference was held at Newington Children's Hospital. This program was initiated under the auspices of Burr H. Curtis, M.D., now Medical Director Emeritus, to provide orthopaedic surgeons and interested allied specialists a program of continuing medical education highlighting recent advances in pediatric orthopaedics.

Newington Children's Hospital has served as a major resource, not only for the care of children with acute and chronic musculoskeletal problems, but as a teaching hospital providing a core curriculum of pediatric orthopaedics for residents from the programs at the University of Connecticut School of Medicine, Dartmouth-Hitchcock Medical Center, and Yale University School of Medicine. Each year's program includes the best papers of residents' basic research and clinical projects for presentation by the orthopaedic staff. Newington Children's Hospital is accredited for postgraduate fellowship in pediatric orthopaedics; we have been fortunate to have frequent presentations by graduate fellows from past years as exemplified this year by Dr. Walter Greene's study of equinovarus deformity of the foot in cerebral palsy. With the virtual elimination of infectious poliomyelitis from both American continents, the field of pediatric orthopaedics has become deeply involved in the treatment of other neuromuscular disorders as indicated in the accompanying abstracts.

On behalf of the medical staff at Newington Children's Hospital, we are pleased to present these abstracts demonstrating the clinical activities of the Department of Orthopaedics at Newington Children's Hospital.

Treatment of Developmental Dysplasia of the Hip in the Child 12 to 36 Months of Age

STEPHEN CURTIN, M.D. AND PETER A. DELUCA, M.D.
Department of Orthopaedic Surgery and Rehabilitation, Yale University School of Medicine,
and Department of Orthopaedic Surgery, Newington Children's Hospital

The treatment of developmental dysplasia of the hip (DDH) in the older child continues to be problematic and fraught with controversy. Thirty-eight developmentally dislocated hips in 33 children between the ages of 12 and 36 months at initial evaluation were treated at our institution between 1972 and 1989. Treatment included a course of traction (87%) followed by arthrographic evaluation and attempted closed reduction under general anesthesia (89%). Interposition of the limbus or other soft tissue blocks to concentric reduction by arthrography were generally felt to be indications for open reduction. Clinical and radiographic evaluation of acetabular dysplasia and femoral anteversion determined whether pelvic or femoral osteotomies were to be performed with the reduction.

Thirty-one hips (81%) were treated with primary open reduction, 20 of which also received primary femoral derotational osteotomy and two of which received primary Salter innominate osteotomy. Seven hips (18%) were treated initially with closed reduction, with one receiving a combined innominate and femoral osteotomy and another receiving a femoral osteotomy. Subsequently, 25 of the 38 hips (66%) underwent at least one secondary procedure, with a total of 38 secondary pelvic or femoral osteotomies being performed.

At an average of eight years of follow-up (range two to 16 years), 18 hips were rated as Class I, 10 as Class II, six as Class III, and three as Class IV according to the classification system of Severin for overall radiographic outcome. One hip could not be classified because of recent surgery. Four hips (10.5%) developed avascular necrosis.

Multiple procedures were required to treat this age group effectively. We found that femoral derotational osteotomy performed at initial reduction significantly decreased the number of subsequent operations and may be associated with a lower incidence of avascular necrosis.

Arthrography was useful in distinguishing two patterns of DDH and guiding treatment, and we recommend its continued use. Longer follow-up is needed as initial good results may deteriorate over time.

Scoliosis Surgery: A Risk Factor for Cholelithiasis?

JONATHAN E. FULLER, M.D., JOHN V. BANTA, M.D., L. CHRISTOPER FOLEY, M.D.,
M.B. OZONOFF, M.D., AND JEFFREY HYAMS, M.D.
Departments of Orthopaedic Surgery, Dartmouth School of Medicine and Newington
Children's Hospital; Department of Radiology, Newington Children's Hospital;
and Department of Pediatric Gastroenterology, Hartford Hospital

Scoliosis surgery has been associated with pancreatitis and acalculous cholecystitis and also has been implicated as a risk factor for cholelithiasis (Teele et al, 1987).

A prospective study of 36 children and adolescents undergoing scoliosis surgery was performed using ultrasound to determine the incidence of cholelithiasis following spine surgery. The patients underwent abdominal ultrasound preoperatively, immediately postoperatively, and then at six, 12, and 24 months following surgery. Final ultrasound exams were done 24 to 60 months following surgery. Four of 36 patients (11.1%) (one male, three females) developed gallstones. The onset of cholelithiasis in three patients was more than six, 12, and 24 months after surgery in each. Factors compared in the patients with and without gallstones included: estimated blood loss, amount of blood transfusion, duration of immobilization, duration of fasting after surgery, and past medical history. Those with gallstones were not different from those without with respect to 14 factors. The only abnormal laboratory values seen in the study sample were related to pancreatitis in four out of 44 patients, as previously reported by Leichtner et al, 1991.

It is concluded that scoliosis surgery is not a risk factor for gallstones because of the delay in onset of cholelithiasis after surgery. Furthermore, the prevalence of gallstones in this study is not greater than that reported in historical control studies of European young adults.

Teele RL, Nussbaum AR, Wyly JB, Allred, EN, Emans J: Cholelithiasis after spinal fusion for scoliosis in children. *J Pediatr* 1987;111:857-60.

Leichtner AM, Banta JV, Etienne N, Schwartz AN, Renshaw TS, Solari LD, Ascione J, Hyams JS: Pancreatitis following scoliosis surgery in children and young adults. *J Pediatr Orthop* 1991; 11: 594-8.

Vertebral Anomalies Associated with Spinal Dysraphism

JOHN T. GORCZYCA, M.D. AND JOHN BANTA, M.D.
Departments of Orthopaedic Surgery,
Dartmouth School of Medicine and Newington Children's Hospital

A retrospective review of 454 patients with spinal dysraphism treated at Newington Children's Hospital was performed to study the incidence and treatment of associated vertebral anomalies. Eighty-one patients (18%) had one or more types of vertebral anomalies, including congenital scoliosis in 52 (11%), congenital kyphosis in 32 (7.0%), and sacral agenesis in 12 (2.6%).

The type of spinal dysraphism present was myelomeningocele in 67 patients, diastematomyelia in 11 patients, and lipomeningocele in nine patients. Five of the six patients with diastematomyelia had myelomeningocele and one had lipomeningocele.

Congenital kyphosis was the vertebral anomaly that most commonly required spinal fusion. Of 32 patients with congenital kyphosis, all had significant progression of their kyphosis and 24 already had spinal fusions.

Multiple vertebral anomalies were the most common type of congenital scoliosis and were present in 19 of the 81 patients. Multiple vertebral anomalies in the thoracolumbar location were the anomalies most likely to have progressive scoliosis. All five patients with multiple anomalies at the thoracolumbar spine had significant curve progression and four underwent fusion.

Eight patients had early limited fusion of their congenital scoliosis between one and seven years of age. Two have not needed second surgical fusions, two have had second fusions that were successfully postponed until after completion of spinal growth, and four have not had second procedures but have not yet completed spinal growth. In a young child with spinal dysraphism and progression of an associated congenital scoliosis, early limited fusion to arrest curve progression is optimal, even if it entails a later second procedure for paralytic deformity.

Split Anterior Tibialis Tendon Transfer (SPLATT) for Equinovarus in Cerebral Palsy

WALTER B. GREENE, M.D.
University of North Carolina School of Medicine

Eighteen patients with cerebral palsy who underwent a SPLATT procedure and who had at least two years follow-up were evaluated. Age at surgery averaged 9.6 years and follow-up averaged 5.7 years. Indications for the SPLATT included: (1) dynamic hindfoot varus, (2) varus that developed during the swing phase, and (3) a positive confusion test (overpull of the anterior tibialis when the seated patient lifts the flexed leg against resistance). Concomitant surgery included fractional lengthening of the posterior tibialis in 12 patients and tendo-Achillis lengthening in 12.

A good result was defined as heel in neutral with slight valgus at initiation of stance, ankle dorsiflexion ≥ neutral, no abnormal calluses, and normal shoe wear pattern. A fair result had mild valgus at initiation of stance and 5-10° heel valgus with the patient standing. A poor result was defined as recurrent varus of >10° or an ankle in > 10° equinus.

Both limbs of the split transfer functioned during walking in all patients. Results were classified as good in 15, fair in three, and there were no poor results. None of the fair results required repeat surgery. One patient who had a good result developed a ganglion cyst at the tendon transfer site that required excision.

Split transfer of the anterior tibialis tendon augments eversion power enough to correct dynamic heel varus in children with cerebral palsy who demonstrate overactivity of the anterior tibialis muscle. This procedure has a high percentage of good results. Although not required in our patients, reversal or modification of the SPLATT could easily be done and is another advantage of this operation.

A Study of Hardware Removal in the Pediatric Population

MICHAEL A. MIRANDA, M.D. PETER A. DELUCA, M.D.,
AND THOMAS S. RENSHAW, M.D.
Departments of Orthopaedic Surgery, University of Connecticut School of Medicine
and Newington Children's Hospital

Removal of hardware is a routine part of the treatment protocol in the pediatric population. We performed a retrospective review of 196 cases of hardware removal at Newington Children's Hospital to evaluate the morbidity and cost of this procedure. Patients included in the study met the following criteria: 20 years of age or less, hardware removal performed in the main operating room, and follow-up of at least two years.

Intra-, pre-, and postoperative records were reviewed. The average follow-up was 56 months. The most common indication for hardware removal was uneventful healing (79%). Coordination of hardware removal with another surgical procedure was the second most common indication (14%). All other indications occurred with an incidence of less than 5%. The overall complication rate was 6.6%, including an infection rate of 3%. No statistically significant predisposition by diagnosis could be detected. All complications occurred after uneventful healing. In 5.8% of the patients, the operative time and estimated blood loss were higher for hardware removal than for implantation. Again, no predisposition for diagnostic groups was detected. Grouping of complications with intraoperative difficulties yielded a total "problem" rate of 12.4%. The average cost of outpatient pin removal was $2,374. The cost of plate removal with overnight stay was $3,661.

This is the largest study of hardware removal reported to date and although our complication rate was acceptable, the overall morbidity and cost of hardware removal were not insignificant. Based on our findings and a critical review of the literature, removal of internal fixation should be justified on an individual basis.

Torsional Malalignment Syndrome

LOEL Z. PAYNE, M.D. AND PETER A. DELUCA, M.D.
Department of Orthopaedic Surgery and Rehabilitation, Yale University School of Medicine,
and Department of Orthopaedic Surgery, Newington Children's Hospital

Torsional malalignment of the patellofemoral joint from femoral anteversion and external tibial torsion has been clinically described but never documented. We report in this study a group of seven adolescent patients (12 extremities) with patellofemoral pain who had severe femoral anteversion and a normal or external thigh-foot angle. These patients are contrasted to a similar group of eight adolescent patients (15 extremities) who have severe femoral anteversion and a normal-to-internal thigh-foot angle but do not have patellofemoral pain. The group with an external thigh-foot angle had an increased average Q angle, and gait laboratory analysis revealed an increased eccentric quadriceps contracture during push-off in the patients with pain. Derotational osteotomy of the deformity in five patients (eight extremities) relieved the knee symptoms in seven of the eight lower extremities. Gait analysis of symptomatic patients with torsional malalignment syndrome may be helpful in defining spastic pathologic patterns.

Preliminary Traction in the Treatment of Congenital Dislocation of the Hip

ROBERT QUINN, M.D., PETER DELUCA, M.D., AND THOMAS RENSHAW, M.D.
Departments of Orthopaedic Surgery, University of Connecticut School of Medicine
and Newington Children's Hospital

The benefit of preliminary traction in the treatment of congenital dislocation of the hip has not been clearly demonstrated. We retrospectively analyzed the results of treatment of 90 dislocated hips in 72 patients, all of whom had received a regimented program of preliminary traction. Patterns of dislocation were analyzed using both plain radiographs and arthrograms. Following a three-week course of traction, patients underwent attempted closed reduction. Fifty-two hips (58%) were managed successfully by closed reduction, while 38 hips (42%) required primary open reduction. Significant avascular necrosis developed in five hips.

Our results were compared to a recently published series in which preliminary traction was not utilized (Kahle et al, 1990). Chi-square analysis revealed no significant difference in either the rate of successful closed reduction or the incidence of avascular necrosis. On the basis of presentation radiographs and arthrograms, we were unable to identify a subgroup of patients that clearly benefitted from the use of traction.

On the basis of our results, we were unable to demonstrate a beneficial role of preliminary traction in the treatment of congenital dislocation of the hip.

Kahle W K, Anderson M B, Alpert J, Stevens P M, and Coleman S S: The value of preliminary traction in the treatment of congenital dislocation of the hip. *J Bone Joint Surg* 1990; 72-A:1043-7.

Computed Tomography (CT) Scan Assessment of Reduction in Developmental Dislocation of the Hip (DDH)

BRIAN G. SMITH, M.D., JAMES R. KASSER, M.D., LLOYD A. HEY, M.D.,
AND MICHAEL B. MILLIS, M.D.
Department of Orthopaedic Surgery, Children's Hospital, Boston, Massachusetts

Over a six-year period from 1985 to 1991, 71 infants underwent closed reduction for DDH. Fifty-four had postoperative CT scans in spica casts for assessment of adequacy of reduction. Twenty scans representing 40 hips were randomly selected for evaluation by three observers, consisting of two senior pediatric orthopaedists and one pediatric orthopaedic fellow. Raters measured eight previously described characteristics of acetabular and femoral anatomy on the CT scans. In addition, a measurement determined by the position of the femoral neck relative to a line derived from the pubic rami, a modified Shenton's line, was also evaluated. Pelvic width on a trans-triradiate baseline was also measured. Mean and standard deviation differences as well as Pearson and Spearman reliability indices were calculated for each of the 10 measured parameters.

Measurement of acetabular anteversion, anteroposterior femoral displacement, Shenton's line, and pelvic width were reliable among raters with Pearson correlation coefficient r values > .75. Test-retest reliability by one rater existed for all of the above measurements and the hip abduction angle as well. Indices of acetabular morphology could not be reliably measured either within or among raters, with anterior acetabular and axial acetabular indices having the least inter-observer agreement (Pearson r values = < .5).

Computed tomography has been identified as a useful way to assess the adequacy of closed reduction of DDH postoperatively in infants in spica casts. In addition, acetabular morphology on CT scans in children with hip instability has also recently been described. Rater reliability of these CT scan measurements on infants following closed reduction for DDH has not been described in the literature. This study demonstrates that measurement of acetabular morphology of CT scans in infants with DDH cannot be reliably measured. Factors that contribute to the inaccuracy of acetabular measurements include the small size and relative skeletal immaturity of the infant's pelvis. Measurements of hip abduction angle, acetabular anteversion, anteroposterior femoral head displacement, and a modified Shenton's line show good statistical correlation among raters. The reliability of these anatomic parameters measured on postoperative CT scans in infants in spica casts following closed reduction for DDH permits the meaningful comparison of these CT scan findings among different patients. Further evaluation of these CT scan measurements for their prognostic significance in predicting maintenance of reduction and likelihood of open reduction or subsequent operative intervention in children with DDH is also merited.

Buckley SL, Sponseller PD, Magid D: The acetabulum in congenital and neuromuscular hip instability. *J Pediatr Orthop* 1991;11:498-501.

Recognition and Management of Atypical Idiopathic Clubfoot

VINCENT J. TURCO, M.D.
St. Francis Hospital and Medical Center

This type of clubfoot constitutes a very small percentage of congenital clubfeet. At birth, the only apparent abnormality is a clubfoot without stigmata of neurological or genetic syndrome.

The response to treatment, especially surgery, is unlike common idiopathic clubfoot. Early surgery usually results in a severe, "overcorrected" flatfoot deformity.

In a retrospective study, the author identified the following characteristic findings that raise a high degree of suspicion.

1. Hypotonia, "loose jointed" delayed development is the most common finding.
2. The Achilles tendon inserts laterally on the calcaneus unlike the usual medial insertion found in the common idiopathic clubfoot.
3. Transverse skin creases in the area of the tendo-Achillis insertion are absent in the common clubfoot. Fine skin creases may be seen after two to three months of manipulation.
4. Contralateral opposite deformity, eg, calcaneovalgus.
5. Rocker-bottom deformity occurs quite frequently at two to three months.
6. Atypical clubfeet are likely to be longer and more flexible, with a long first ray.

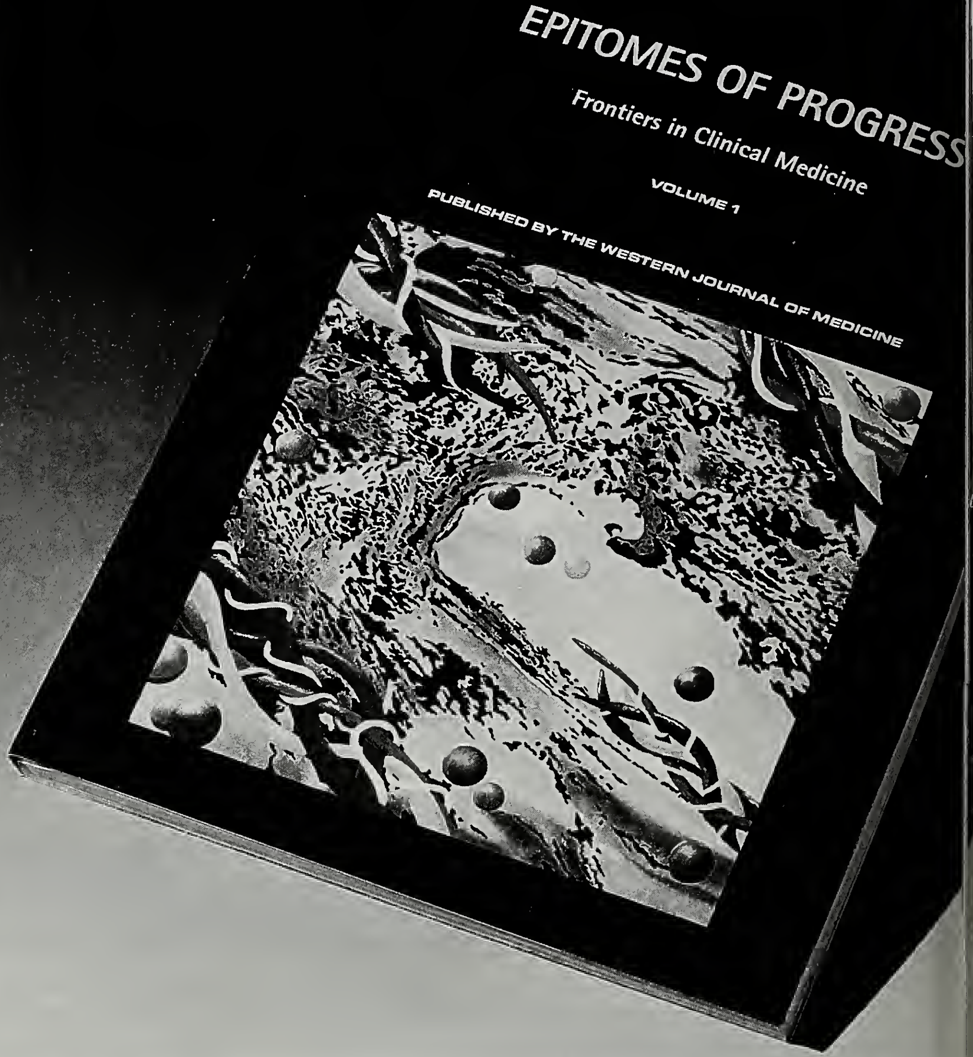
Treatment

Nonoperative treatment is recommended for suspected atypical idiopathic clubfeet. Serial manipulation and casting are started in the newborn nursery. The initial response to treatment appears to be quite favorable. The suspicious characteristic findings usually become evident by two to three months. Casting is discontinued and a Denis-Browne bar and passive stretching are instituted.

Good results have been attained with this nonoperative management. Recognition is imperative in order to avoid severe, overcorrected flatfoot deformity, which tends to occur after early surgery.

epitome

i-'pit-ə-mē\ *n* [L, fr. Gk *epitomē,* fr. *epitemnein* to cut short, fr. *epi-* + *temnein* to cut — more at TOME] (1520) **1 a** : a summary of a written work **b** : a brief presentation or statement of something **2** : a typical or ideal example: EMBODIMENT (the British monarchy itself is the ~ of tradition — Richard Joseph) **3** : Plural — *Epit-o-mes* — A regular FEATURE of *The Western Journal of Medicine,* now available in a single, bound volume.


EPITOMES OF PROGRESS
Frontiers in Clinical Medicine
VOLUME 1
PUBLISHED BY THE WESTERN JOURNAL OF MEDICINE

Each set of **EPITOMES** is a concise and clear review of the important advances in a medical specialty, chosen and written by experts in that specialty. Now, for the first time, a special bound volume of **EPITOMES** is available. For only $19.95 you can have all the latest advances in 24 different specialties in one attractively designed volume. An excellent reference for your library. A useful gift. Hurry! Supplies are limited. Order a copy today.

YES, SEND A SPECIAL BOUND EDITION OF **EPITOMES** TO:

The Western Journal of Medicine

☐ Yes, send me ____ copy(ies) of the Epitomes book at $19.95 each.

☐ I want to receive Epitomes every month. Please enter my one-year subscription to *WJM* for only $40.00.

For additional information, call (415) 882-5179.
Please mail this coupon with enclosed payment to:
Circulation Department
P.O. Box 7602, San Francisco, California 94120-7602

Name ____________________
Street ____________________
City ____________________
State ____________ Zip ____________
☐ Check enclosed Charge my: ☐ VISA ☐ MasterCard
Card Number ____________________
Expiration Date ____________________
Signature ____________________

Important Advances in Clinical Medicine

Ophthalmology

The Council on Scientific Affairs of the California Medical Association presents the following inventory of items of progress in ophthalmology. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome, and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, researchers, and scholars to stay abreast of these items of progress in ophthalmology that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Ophthalmology of the California Medical Association, and the summaries were prepared under its direction.

Medical Treatment of Retinal Infections in Patients With AIDS

CYTOMEGALOVIRUS (CMV) retinopathy and other retinal infections are among the most feared complications of the acquired immunodeficiency syndrome (AIDS). There are now two drugs approved by the Food and Drug Administration for the treatment of CMV retinopathy: ganciclovir sodium and foscarnet. Both are administered intravenously in a similar manner. Initially patients are given an "induction" course of high doses to suppress infection, followed by a life-long "maintenance" regimen to prevent disease reactivation and progression, as neither drug eliminates virus from the eye. The safety and efficacy of these drugs were recently compared in a multicenter clinical trial sponsored by the National Institutes of Health. There were no differences in major ophthalmic end points such as final visual acuity, but the protocol was suspended early when it was determined that patients treated with foscarnet survived several months longer than patients treated with ganciclovir. The cause for the differential survival rates could not be determined. Although this finding could possibly be related to the drugs themselves, it could also have been related to other, uncontrolled factors, such as a differential use of antiretroviral drugs. Both drugs have serious systemic side effects; ganciclovir is a bone marrow suppressant, and foscarnet causes renal damage. Despite the increased survival associated with the use of foscarnet, patients did not tolerate it as well as ganciclovir. Because both drugs protect against loss of vision, patients and their primary care providers should participate in the decision for choosing which drug to use, based on nonophthalmic factors such as cost (foscarnet is more expensive currently than ganciclovir), tolerance, the risk of side effects, and perceived survival benefit.

After ganciclovir became available, the intravitreal administration of the drug was studied as an alternative route for patients with drug-induced neutropenia. Interest in intravitreal ganciclovir has waned for several reasons: Neutropenia can now be treated with the concurrent use of leukocyte growth factors sargramostim (granulocyte-macrophage colony-stimulating factor [GM-CSF]) and filgrastim (granulocyte colony-stimulating factor [G-CSF]); patients can be switched from ganciclovir to foscarnet or vice versa as toxic reactions develop. Intravitreal injections have their own associated risks, such as vitreous hemorrhage and bacterial endophthalmitis. This therapy is now usually reserved for patients who cannot tolerate any parenteral drug.

Reprinted from *The Western Journal of Medicine*, October 1992.

In addition to CMV retinopathy, there are other, less common but equally serious, human immunodeficiency virus-related retinal infections. At least a dozen pathogens have been reported, but varicella-zoster virus (VZV) retinitis and ocular toxoplasmosis are the most common. Varicella-zoster virus retinitis is a rapidly progressive necrotizing infection, and patients should be treated immediately with high-dose intravenous acyclovir sodium; an initial ten-day course is usually recommended. A decision to continue therapy with either intravenous or oral acyclovir is currently based on the patient's response to treatment and the appearance of retinal lesions. The need for long-term maintenance therapy to prevent recurrent VZV retinitis is a subject of controversy. Because many patients are left with poor vision despite treatment, the concurrent use of several intravenous antiviral drugs is being investigated.

Ocular toxoplasmosis is treated with a combination of pyrimethamine and sulfadiazine or clindamycin. Although retinal lesions respond well to this treatment, they can reactivate unless patients are maintained on continuous low-dose therapy with at least one drug. Clindamycin is used most commonly for this purpose because of pyrimethamine-associated bone marrow suppression and the frequency of allergic reactions to sulfonamides in AIDS patients. Several new drugs are also under study for the treatment of *Toxoplasma gondii* infections.

Unfortunately, serologic tests cannot confirm these diagnoses because of the high prevalence of antibodies to CMV, VZV, and *T gondii* in the general population, and retinal biopsies are too risky for diagnostic use in most situations. Ophthalmologists distinguish among them on the basis of subtle differences in lesion appearance, distribution, course, and inflammatory signs. Primary care providers should suspect infections other than CMV retinopathy if an eye becomes red and painful; CMV infection does not cause sufficient inflammation to produce those signs, but toxoplasmosis, syphilis, and fungal infections can.

Because retinal tissue destroyed by infection cannot be regenerated, visual results depend on early diagnosis. Patients should be encouraged to seek immediate ophthalmologic evaluation if they have blurring, visual field defects (blind spots), or multiple floating spots before one or both eyes. With accurate diagnosis and aggressive therapy, many AIDS-related retinal infections can be controlled. Therapies continue to evolve as new drugs become available and effective treatment regimens are identified.

Gary N. Holland, M.D.
Los Angeles, California

REFERENCES

Hardy WD: Combined ganciclovir and granulocyte-macrophage colony-stimulating factor in the treatment of cytomegalovirus retinitis in AIDS patients: Rationale for and preliminary results from a phase 11 randomized trial (ACTG 073), *In* Spector SA (Ed): Ganciclovir Therapy for Cytomegalovirus Infection. New York, NY, Marcel Dekker, 1991, pp 197-213.

Holland GN, Engstrom RE Jr, Glasgow BJ, et al: Ocular toxoplasmosis in patients with the acquired immunodeficiency syndrome. *Am I Ophthalmol* 1988; 106:653-67.

Margolis TP, Lowder CY, Holland GN, et al: Varicella-zoster virus retinitis in patients with the acquired immunodeficiency syndrome. *Am J Ophthalmol* 1991; 112:119-31.

Studies of Ocular Complications of AIDS Research Group, in collaboration with the AIDS Clinical Trials Group: Mortality in patients with the acquired immunodeficiency syndrome treated with either foscarnet or ganciclovir for cytomegalovirus retinitis. *N Engl J Med* 1992; 326:213-20.

Photorefractive Keratectomy

PHOTOREFRACTIVE keratectomy is a surgical procedure to correct nearsightedness (myopia), farsightedness (hyperopia), and astigmatism. The procedure involves recontouring the anterior surface of the cornea with an excimer laser that uses ultraviolet light (193 nm). Each laser pulse ablates 0.2 to 0.3 µm of corneal tissue, with almost no thermal or other injury produced in the immediately adjacent tissue. The anterior cornea is reshaped by ablating small amounts of tissue from the corneal surface; to flatten the cornea, more tissue is removed centrally than is removed from the periphery, and corneal steepening is achieved by removing little or no tissue centrally and progressively more tissue towards the corneal periphery. The end result is that a central round area of the cornea measuring between 4 and 6 mm in diameter and overlying the pupil is treated, with a maximum depth of ablation of about 10% of corneal thickness. Clinical trials of photorefractive keratectomy are under way in the United States at about 30 centers, and the Food and Drug Administration (FDA) is carefully controlling the number of patients enrolled in these studies. The FDA has determined that five-year follow-up will be evaluated before deciding whether or not to approve this procedure for the correction of refractive errors in otherwise healthy eyes. In Europe, Canada, and Asia, where governmental regulation is less restrictive, many lasers are in place and more than 10,000 eyes have been treated.

Data from the clinical sites in the United States and reports from abroad show that, in the short run, the procedure appears to be reasonably safe and effective for the correction of low-to-moderate myopias. Beyond 6 diopters, partial or even total regression of the effect is common, resulting in poor predictability of ultimate results. Because 90% of nearsighted Americans have refractive errors of 5 D or less, most patients are candidates who could expect a favorable result if they need any correction beyond spectacles or contact lenses. Clinical follow-up on a large number of patients has been limited to about two years, so long-term safety and stability remain to be determined. In the short term, a superficial corneal haze is typical after the procedure, but this generally decreases in intensity over time. A few patients have measurable decreases in visual acuity or contrast sensitivity for several weeks after the operation; a persistent decrease in visual acuity due to substantial corneal haze occurred in about 2% of patients in one center in Europe. Although most treatments have been performed for myopia, recent studies suggest this laser can be effective also for the treatment of astigmatism.

Photorefractive keratectomy is to be distinguished from radial keratotomy, a procedure that has been done in the United States since 1978 to correct nearsightedness and astigmatism. Radial keratotomy involves creating incisions deep (90% depth or greater) into the corneal stroma. Four, eight, or more incisions are created, starting in the paracentral cornea and extending radially to the corneal periphery. A large multicenter National Eye Institute-sponsored clinical trial, the Prospective Evaluation of Radial Keratotomy (PERK) Study, was carried out in the early and mid-1980s to define the safety and effectiveness of this procedure. Vision-threatening complications are rare after radial keratotomy, but predictability is somewhat limited; about two thirds of the patients in the PERK study had adequate vision without spectacles or contact lenses after the operation.

In addition to correcting refractive error, the excimer laser is also being used, as an alternative to corneal transplantation, to remove anterior corneal opacities. This procedure, called phototherapeutic keratectomy, has been largely successful to date, and the FDA is currently considering granting approval of the laser for this indication. Should approval be granted, this procedure will provide an outpatient, cost-saving surgical alternative to about 10% of the 40,000 patients who undergo corneal transplant each year in the United

Peter J. McDonnell, M.D.
Los Angeles, California

REFERENCES

McDonald MB, Frantz JM, Klyce SD, et al: One-year refractive results of central photorefractive keratectomy for myopia in the nonhuman primate cornea. *Arch Ophthalmol* 1990; 108:40-7.

McDonnell PJ, Moreira H, Clapham TN, D'Arcy J, Munnerlyn CR: Photorefractive keratectomy for astigmatism: Initial clinical results. *Arch Ophthalmol* 1991; 109: 1370-3.

Seiler T, Wollensak J: Myopic photorefractive keratectomy with the excimer laser: One-year follow-up. *Ophthalmology* 1991; 98: 1156-63.

Sher NA, Bowers RA, Zabel RW, et al: Clinical use of the 193-nm excimer laser in the treatment of corneal scars. *Arch Ophthalmol* 1991; 109:491-8.

Macular Surgery

THE application of vitreoretinal microsurgical techniques to disorders of the macula, the central retinal visual area, has become one of the most exciting and rapidly developing areas in ophthalmology. Until recently the primary indication for a macular operation has been the treatment of avascular membranes that may develop on the surface of the macula, descriptively termed "macular pucker." Since the late 1970s the surgical removal of epimacular membranes has improved the vision in about 80% of patients.

The development of idiopathic full-thickness macular holes is a common cause of central vision loss, typically affecting older women, and occurring in both eyes in 10% to 20% of patients. A new hypothesis implicated centrifugal traction of the vitreous parallel to the retinal surface at the vitreomacular interface (tangential traction) in the pathogenesis of idiopathic full-thickness macular holes. This prompted the report that the surgical treatment of macular holes has met with stunning yet widely corroborated success.

Previously considered to have an irreparable condition, 42% of patients had improvement in their vision, with 19% returning to near-normal visual function. Continuing refinements in surgical techniques suggest that substantially better success rates may be expected. Surgical prophylaxis for impending macular holes, when visual symptoms and clinical signs may be subtle yet reversible, is the subject of an ongoing multicenter, prospective clinical trial.

Successful macular hole operations have cast doubt on previous concepts of vitreomacular pathophysiology based on clinical evaluation. Detachment of the vitreous from the surface of the macula is a common and typically benign age-related event. It is now recognized, however, that the exact relationship of the posterior vitreous to the macula can often be determined only at the time of surgical intervention. The most careful preoperative evaluation has frequently been found at the time of a macular operation to be misleading and unreliable, either not recognizing or underestimating the role of the vitreous in the pathologic process. Other disorders, such as loss of vision due to anteroposterior vitreomacular traction caused by incomplete vitreous detachment from the macula, have been recognized and shown to be responsive to surgical intervention. Indeed, it is increasingly appreciated that macular pucker, as well as tangential and anteroposterior vitreomacular traction, may occur alone or in concert, even superimposed on other macular disease, causing or contributing to loss of central vision that may be amenable to surgical intervention.

The development of neovascular membranes beneath the macula is an important cause of central vision loss in a multitude of disorders, particularly the presumed ocular histoplasmosis syndrome and, more commonly, age-related macular degeneration. Recently the surgical removal of submacular neovascular membranes complicating presumed ocular histoplasmosis syndrome and age-related macular degeneration has been reported with encouraging, occasionally remarkable, results. Such developments have ushered in a new era in vitreoretinal surgical treatment by revolutionizing our understanding of clinical vitreomacular pathophysiology and expanding the application of current microsurgical techniques to macular diseases until only recently unrecognized, misunderstood, or considered irreparable.

Jeffrey K. Luttrull, M.D.
Ventura, California

REFERENCES

Gass JDM: Idiopathic senile macular hole—Its early stages and pathogenesis. *Arch Ophthalmol* 1988; 106:629-39.

Kelly NE, Wendel RT: Vitreous surgery for idiopathic macular holes. *Arch Ophthalmol* 1991 109:654-9.

Lambert HM, Capone A, Aaberg TM, Sternberg P, Mandell BA, Lopez PF: Surgical excision of subfoveal neovascular membranes in age-related macular degeneration. *Am J Ophthalmol* 1992; 113: 257-62.

Lewis H, Abrams GW, Blumencranz MS, Campo RV: Vitrectomy for diabetic macular traction and edema associated with posterior hyaloidal traction. *Ophthalmology* 1992; 99:753-9.

Thomas MA, Kaplan HJ: Surgical removal of subfoveal neovascularization in the presumed ocular histoplasmosis syndrome. *Am J Ophthalmol* 1991; 111:1-7.

Laser Treatment of Diabetic Retinopathy

DIABETES mellitus is the leading cause of blindness in working-age Americans and accounts for 8,000 cases of new blindness each year in the United States. “Diabetes 2000” is an ambitious, long-term education project of the American Academy of Ophthalmology, designed to eliminate preventable blindness from cases of diabetes by the year 2000. Ophthalmologists and primary care physicians must share advances to accomplish this goal.

Two distinct forms of diabetes mellitus are recognized— juvenile onset (type I) and adult onset (type II). Furthermore, two distinct forms of diabetic retinopathy that may cause blindness occur—proliferative diabetic retinopathy and diabetic macular edema. Proliferative diabetic retinopathy consists of neovascular tissue that grows on the surface of the retina and into the vitreous cavity. If untreated, bleeding occurs into the vitreous, which then contracts, sometimes resulting in retinal detachment. Diabetic macular edema is caused by incompetent vessels leaking serum and lipid into a poorly perfused central retina. As retinal thickening occurs, photoreceptor function decreases and central vision is compromised. Juvenile-onset diabetes carries a higher risk of severe proliferative retinopathy, but because there are more cases of adult-onset diabetes, the latter group comprises a significant proportion of patients with blindness. In many instances, blindness from diabetes can be prevented by early detection and prompt photocoagulation treatment.

In 1976 a National Eye Institute (NEI)-funded Diabetic Retinopathy Study showed that panretinal scatter photocoagulation reduced the rate of profound loss of vision from severe proliferative diabetic retinopathy by 50%. Results are lasting: Of high-risk patients who had a favorable response to photocoagulation, 80% maintained 20/40 (reading) vision five years after treatment. The effects of photocoagulation on the management of less severe stages of diabetic retinopathy were not known until recently. A second NEI clinical trial showed that early treatment, when compared with observation only, was associated with a small reduction in the incidence of severe vision loss, but five-year rates of loss of vision were low in both the early treatment and observation groups. Therefore, if careful follow-up can be maintained, scatter photocoagulation is not recommended for eyes with mild or moderate nonproliferative diabetic retinopathy. When retinopathy is more severe, scatter photocoagulation should be considered and should not be delayed if the eye has reached the high-risk proliferative stage.

Diabetic macular edema is a more common cause of severe loss of vision than proliferative diabetic retinopathy. For eyes with macular edema, focal macular photocoagulation is the only effective treatment that reduces the risk of moderate visual loss. Treatment should be considered only for eyes with clinically significant macular edema, such as edema that involves or threatens the center of the macula. Focal treatment stabilizes visual acuity, increases the chance of visual improvement, decreases the rate of subsequent loss of vision by decreasing the frequency of persistent macular edema, and causes only minor visual field loss. Timing is therefore important. Treatment should ideally be applied before serious vision loss occurs.

Photocoagulation has been proved to be cost effective. A computer-simulation model was designed to predict the economic effects of the treatment of diabetic retinopathy in juvenile diabetes. Using treatment recommendations provided by these clinical trials, the model predicted a cost savings of $9,571 per year for each new patient diagnosed and appropriately treated. If all patients with diabetes mellitus received appropriate care, the predicted annual savings would exceed $167 million and 79,236 person-years of sight. Unfortunately, it is estimated that 22% of patients with type I diabetes and 40% of those with type II diabetes are not obtaining appropriate eye care. It is less expensive to provide preventive care than to support subsequent disability and its associated personal and social suffering.

Using the results of these clinical trials, a treatment plan for diabetic retinopathy has been designed and proved medically and cost effectively. The trials have shown that most patients with diabetes go blind because they are treated too late. They must be evaluated early to reap the benefits of these studies.

Paul E. Tornambe, M.D.
La Jolla, California

REFERENCES

Early Treatment Diabetic Retinopathy Study Research Group: Early photocoagulation for diabetic retinopathy—ETDRS Report Number 9. *Ophthalmology* 1991; 98(suppl): 766-85.

Early Treatment Diabetic Retinopathy Study Research Group: Early treatment diabetic retinopathy study design and baseline patient characteristic—ETDRS Report Number 7. *Ophthalmology* 1991: 98(suppl):741-53.

Javitt JC, Canner JK, Sommer A: Cost effectiveness of current approaches to the control of retinopathy in type I diabetics. *Ophthalmology* 1989; 96:255-64.

Klein R, Klein BE, Moss SE, Davis MD, DeMets DL: The Wisconsin Epidemiologic Study of Diabetic Retinopathy: X. Four-year incidence and progression of diabetic retinopathy when age at diagnosis is 30 years or more. *Arch Ophthalmol* 1989; 107:244-9.

Vander JF, Duker JS, Benson WE, Brown GC, McNamara JA, Rosenstein RB: Long-term stability and visual outcome after favorable initial response of proliferative diabetic retinopathy to panretinal photocoagulation. *Ophthalmology 1991;* 98:15759.

Retinitis Pigmentosa—New Advances in Ophthalmic Genetics

RETINITIS pigmentosa is both clinically and genetically heterogeneous. It comprises a group of X-linked autosomal recessive and autosomal dominant diseases that affect 50,000 to 100,000 people in the United States, with the incidence ranging from one in 3,000 to one in 7,000. The early symptoms of retinitis pigmentosa include night blindness and a loss of peripheral fields of vision. As the condition progresses, visual acuity and central vision are also affected. Examination of the fundus reveals attenuated retinal vessels, bone-spicule pigment around the periphery of the retina, and occasionally pale optic discs.

Molecular biologic studies have defined the genetic defects for at least some of the persons and families who have autosomal-dominant retinitis pigmentosa. Most of the mutations discovered to date are the result of changes in a single nucleotide in the rhodopsin gene. Different families may carry different mutations, but affected members of the same family carry the same dominantly inherited mutation. These mutations lead to single amino acid changes in the rhodopsin molecule. The first mutation discovered was a C-to-A base change in the 23rd codon of the rhodopsin gene that resulted in a substitution in the 23rd amino acid of histidine for proline. This mutation was denoted as the proline-23-histidine mutation. In a recent report of 161 unrelated patients with autosomal-dominant retinitis pigmentosa who were screened, 26 (24%) were found to carry a point mutation in the rhodopsin gene. This study described 13 different mutations at 12 different amino acid positions. Research is under way to uncover the molecular defects in the rest of these patients by exploring the possibility of defects in other retinal and photoreceptor genes, such as arrestin (also known as S antigen), transducin, peripherin, and others.

Many investigators have attempted to correlate observed genetic heterogeneity with variability in the clinical severity and prognosis of autosomal-dominant retinitis pigmentosa. Initial observations suggested that patients with certain mutations, such as proline-23-histidine, threonine-58-arginine, glycine-182-serine, and threonine-17-methionine, may have better long-term prognoses than others for the retention of good visual acuity and functional peripheral visual fields. In patients with the proline-347-leucine mutation, there is an increased likelihood of more severe functional impairment. The age of onset and severity of the disease, however, may vary substantially among patients with the same mutation.

How can these new discoveries be used to diagnose and treat patients with retinitis pigmentosa? Molecular genetics, in conjunction with a review of family history and careful clinical examination, can in some cases determine whether a person with a family history of autosomal-dominant retinitis pigmentosa is carrying the disease gene while asymptomatic. This information can assist persons in making career and family choices. The application of further clinical and basic research will one day help to decipher the biochemical basis for retinal degeneration in retinitis pigmentosa, which we hope will lead to a rational basis for treating the disorder. Further research is also required to determine the molecular defect in the great majority of persons with dominantly inherited retinitis pigmentosa, as well as in patients with the autosomal recessive and X-linked type. Patients with this disorder optimally should be referred to centers with experience in its diagnosis and treatment and be enrolled in clinical studies.

Vinh Tan Tran, M.D., Ph.D.
Los Angeles, California

REFERENCES

Berson EL, Rosner B, Sandberg MA, Dryja TP: Ocular findings in patients with autosomal dominant retinitis pigmentosa and a rhodopsin gene defect (Pro-23-His). *Arch Ophthalmol* 1991; 109: 92-101.

Dryja TP, McGee TL, Reichel E, et al: A point mutation of the rhodopsin gene in one form of retinitis pigmentosa. *Nature* 1990; 343:364-6.

Fishman GA, Stone EM, Sheffield VC, Gilbert LD, Kimura AE: Ocular findings associated with rhodopsin gene codon 17 and codon 182 transition mutations in dominant retinitis pigmentosa. *Arch Ophthalmol* 1992; 110:54-62.

Sung CH, Davenport CM, Hennessey JC, et al: Rhodopsin mutations in autosomal dominant retinitis pigmentosa. *Proc Natl Acad Sci USA* 1991; 88:6481-5.

Current Therapy for Disorders of the Optic Nerve

OPTIC neuritis is characterized by subacute unilateral loss of central vision in patients younger than 40 years, associated with pain on eye movement, diminished pupillary response, and optic disc edema in about 40% . It may be associated with systemic disorders such as systemic lupus erythematosus and syphilis and may be mimicked by parasellar tumors. Most cases are idiopathic, in which there may be a strong association with multiple sclerosis. The value of corticosteroid therapy in idiopathic optic neuritis has not been proved. Early reports indicated that treatment reduced pain and increased the speed of visual recovery but did not appreciably affect the final level of visual acuity. Most neuro-ophthalmologists have treated only those patients with severe pain or with a definitive need for the rapid recovery of vision, such as patients with severe loss of vision, or only one functional eye. The recent national multicenter clinical trial of systemic corticosteroid use in optic neuritis (Optic Neuritis Treatment Trial) showed that oral prednisone therapy alone had no beneficial effect and was associated with an increased risk of recurrence; study investigators have therefore recommended against its use. An initial course of intravenous methylprednisolone sodium succinate, however, followed by oral prednisone was beneficial in patients with visual acuity worse than 20/40. In patients with more severe vision loss, consideration should be given to this therapy.

Nonarteritic anterior ischemic optic neuropathy, an infarct of the optic nerve head, is the most common cause of acute optic neuropathy in patients older than 50. It presents as acute, painless, unilateral loss of vision associated with impaired pupillary response and optic disc swelling. It typically occurs as a single episode but may progress over a period of weeks in 20% to 30% of cases. Recent studies suggest that optic nerve sheath decompression, a surgical procedure in which cerebrospinal fluid is released through an incision into the subarachnoid space surrounding the optic nerve, may reduce permanent optic nerve damage in the progressive form of nonarteritic anterior ischemic optic neuropathy. The procedure, which theoretically works by reducing intrasheath pressure, allowing for improved blood flow to the optic nerve head, has proved effective in chronic optic disc edema from increased intracranial pressure (most commonly pseudotumor cerebri). Its use in nonarteritic anterior ischemic optic neuropathy, however, is controversial. A recent series of reports indicated notably improved vision after optic nerve sheath decompression in a high percentage of patients, but several investigators have presented data that do not corroborate this success. A multicenter clinical trial, the Ischemic Optic Neuropathy Decompression Trial, has recently been funded by the National Eye Institute to evaluate the effectiveness of this therapy; recruitment of some 300 patients over 18 months was started in 1992.

Anthony C. Arnold, M.D.
Los Angeles, Callfornia

REFERENCES

Beck RW, Cleary PA, Anderson MM Jr, et al: A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. *N Engl J Med* 1992; 326:581-8.

Kelman SE. Elman MJ: Optic nerve sheath decompression for nonarteritic ischemic optic neuropathy improves multiple visual function measurements. *Arch Ophthalmol* 1991; 109:667-71.

Spoor TC, Wilkinson MJ, Ramocki JM: Optic nerve sheath decompression for the treatment of progressive nonarteritic ischemic optic neuropathy. *Am J Ophthalmol* 1991; 724-8.

Graves' Ophthalmopathy

THE diagnosis of Graves' ophthalmopathy is purely clinical and is unrelated to the systemic thyroid state. Although most patients have hyperthyroidism, euthyroid patients or those with hypothyroidism also present with ocular symptoms. Orbital computed tomographic scans are helpful in predicting possible loss of vision but are rarely necessary for the diagnosis of Graves' ophthalmopathy. Although some form of symptomatic ocular involvement is present in at least 31% of patients with Graves' thyroid disease, the ophthalmopathy may occur, both in timing and severity, independent of the thyroid disease.

Data on the natural history of Graves' ophthalmopathy remain scarce. Most investigators agree that the disease is self-limiting and runs a course of exacerbations and spontaneous remissions, becoming stable within 18 to 36 months. Hence, ongoing collaboration among primary care physicians, endocrinologists, and ophthalmologists is paramount for optimal patient care.

There is no known effective treatment to prevent the development of Graves' ophthalmopathy. Our efforts remain directed at palliating symptoms. Managing the thyroid dysfunction in these patients is the first logical step in the treatment of Graves' ophthalmopathy, but the disorder may continue to progress.

When considering treatment, patients can be divided into three major categories: those with active vision-threatening optic neuropathy, those with active nonvision-threatening ophthalmopathy, and those with chronic stable ophthalmopathy. Patients with acute Graves' optic neuropathy require urgent treatment to prevent permanent loss of vision. The longer treatment is delayed, the less adequate visual recovery will be. Corticosteroid therapy is generally the first line of treatment. In patients who cannot tolerate corticosteroids, cyclosporine has been recently suggested as an alternative or adjunct to corticosteroids, but its effects on ophthalmopathy are not well documented and the side effects are potentially serious. For patients who have a recurrence of optic neuropathy with tapering of their corticosteroid regimen or for those who do not respond to or simply cannot tolerate corticosteroid therapy, either orbital decompression or orbital irradiation is indicated. Orbital decompression remains a successful treatment of optic neuropathy, whereas orbital radiotherapy remains controversial because of the risks of dry eye and radiation retinopathy associated with low-dose irradiation. In addition, the risk of inducing tumors is unknown.

For patients with nonvision-threatening acute ophthalmopathy, symptomatic treatment and expectant follow-up are indicated. Artificial tears, lubricants, night-time patching of the eyelids, elevation of the head of the bed, and wrap-around glasses are helpful treatments. No controlled trials have been done that demonstrate the efficacy of corticosteroid therapy or irradiation in altering the natural history of the ophthalmopathy or in improving proptosis or diplopia once the disease has become stable. The risk of corticosteroid use or irradiation must therefore be weighed against the short-term improvement in active inflammation. If steroids are prescribed, they should be given over a course of two to three weeks, with rapid tapering to avoid prolonged treatment even if inflammation recurs.

Once fibrosis has developed in patients with chronic stable thyroid ophthalmopathy, neither corticosteroid nor radiation therapy has a role because active inflammation is not present. For these patients, surgical reconstruction may be considered. Most surgeons agree that the reconstructive operations should be done in sequence with orbital decompression, followed by the correction of double vision, and, finally, an eyelid operation. The surgical procedure must be tailored to the special needs of each patient. Cosmetic orbital decompression in the absence of functional diplopia must be undertaken with caution because diplopia will develop in 30% to 50% of patients after surgical decompression.

An often overlooked yet equally important component of Graves' disease is the psychosocial stress associated with the disease. Graves' disease often has substantial economic and social effects, as patients are commonly in their wage-earning years and, in some cases, are unable to do their jobs because of their visual symptoms. The cosmetic disfigurement produced by ophthalmopathy may cause a reactive depression, placing stress on the family. These patients may be referred to a support group through the National Graves' Disease Foundation (11111-2A San Jose Blvd, Suite 123, Jacksonville, FL 32223).

Denise F. Dudley, M.D.
James C. Orcutt, M.D., Ph.D.
Seattle, Washington

REFERENCES

Garity IA: Treatment of Graves' orbitopathy in 1990. *Keio I Med* 1991; 40:63-71.

Mourits MP, Koorneef L, Wiersinga WM, Prummel MF, Berghout A, van der Gaag R: Critical criteria for the assessment of disease activity in Graves' ophthalmology: A novel approach. *Br J Ophthalmol* 1989; 73:639-44.

Stasior GO, Krohel GB: Pathophysiology, management, and surgical review of thyroid-related ophthalmopathy. *Curr Opin Ophthalmol* 1991; 2:621-8.

Small-Incision Cataract Operation

CATARACT extraction with intraocular lens implantation is currently the most common surgical procedure in the United States. In the past 20 years a revolution has occurred in the techniques and technology of cataract operations. With the earlier "intracapsular" technique, the lens and capsule were removed intact through a large (10 to 14 mm) incision at the edge of the cornea. This large incision required more suturing to close, was more traumatic to the eye, and often resulted in postoperative astigmatism. When studies showed that leaving the posterior lens capsule intact would reduce the risk of postoperative macular edema and retinal detachment, most surgeons adapted an "extracapsular" technique wherein the anterior lens capsule was opened and the lens nucleus manipulated forward and removed from the eye through a large incision.

The introduction of phacoemulsification in the 1970s began a sequence of innovations that made small-incision operations possible in the late 1980s. Phacoemulsification is the process of concentrating ultrasonic energy through a handpiece to the lens nucleus. The nucleus is fragmented into fine particles that are aspirated from the eye. The lens can be removed entirely through a 3-mm incision. To implant an intraocular lens in the eye, however, the wound still had to be extended to 6 to 7 mm.

Other innovations soon followed. Viscoelastic agents provided a clear, viscous material that could coat and protect the corneal endothelium from the ultrasonic energy, making the cataract operation safer for the cornea. These agents also helped maintain the shape of the anterior chamber and allowed for excellent visibility during procedures. Capsulorhexis, a technique for making a controlled round or oval tear in the anterior capsule, preserved the strength and elasticity of the remaining lens capsule. This technique made phacoemulsification safer and intraocular lens placement more consistent. New methods of dissecting the lens nucleus and separating the layers of lens cortex using cannulas and irrigation were introduced, which allowed easier access to the nucleus and a greater margin of safety during phacoemulsification. Intraocular lens designs were tailored for smaller incisions. Lenses were designed to fit through a 5-mm incision; foldable silicone lenses capable of insertion through a 4-mm incision were also introduced. Wound designs were devised that are self-sealing, making "single-stitch" and "no-stitch" operations possible.

Small-incision techniques are being adapted by a growing percentage of ophthalmologists. Recent studies have shown that the main advantages of small-incision cataract operations are less trauma to ocular tissues, less immediate postoperative inflammation, less postoperative astigmatism, and more rapid visual rehabilitation.

Richard H. Keates, M.D.
Kevin H. Merkley, M.D.
Irvine, California

REFERENCES

Brint SF, Ostrick DM, Bryan JE: Keratometric cylinder and visual performance following phacoemulsification and implantation with silicone small-incision or poly(methyl methacrylate) intraocular lenses. *J Cataract Refract Surg* 1991; 17:32-6.

Gills JP, Sanders DR: Use of small incisions to control induced astigmatism and inflammation following cataract surgery. *J Cataract Refract Surg* 1991; 17(suppl):7404.

Siepser SB: Sutureless cataract surgery with radial transverse incision. *J Cataract Refract Surg* 1991; 17(suppl):716-8.

Steinert RF, Brint SF, White SM, Fine IH: Astigmatism after small incision cataract surgery—A prospective, randomized, multlcenter comparison of 4- and 6.5-mm incisions. *Ophthalmology* 1991; 98:417-23. [discussion 423-4]

ADVISORY PANEL TO THE SECTION ON OPHTHALMOLOGY

Bernice Z. Brown, M.D.
Advisory Panel Chair
CMA Council on Scientific Affairs Representative
Los Angeles

Yossi Sidikaro, M.D.
CMA Section Chair
Beverly Hills

Bernd M. Kutzscher, M.D.
CMA Section Secretary
Daly City

Kenneth R. Diddie, M.D.
CMA Section Assistant Secretary
Section Editor
Santa Monica

David L. Wilkins, M.D.
Loma Linda University

Peter Egbert, M.D.
Stanford University

John Keltner, M.D.
University of California
Davis

Richard H. Keates, M.D.
University of California, Irvine

Bradley R. Straatsma, M.D.
University of California
Los Angeles

Stuart I. Brown, M.D.
University of California
San Diego

Steven G. Kramer, M.D.
University of California
San Francisco

Stephen J. Ryan, Jr., M.D.
University of Southern California

Donald N. Schwartz, M.D.
California Association of
Ophthalmology
Long Beach

Philip L. Levy, M.D.
California Association of
Ophthalmology
Sacramento

Robert C. Black, M.D.
California Association of
Ophthalmology
Fremont

Robert L. Stamper, M.D.
California Pacific Medical Center
San Francisco

Ray T. Oyakawa, M.D.
White Memorial Medical Center
Los Angeles

John Richards
Medical Student Representative
University of California, Davis

Practice Locally, Search Globally

FAITH MCGRATH, M.L.S. AND NICHOLAS G. TOMAIUOLO, M.L.S.

ABSTRACT—The National Library of Medicine introduced the Grateful Med software package for accessing its databases over six years ago. Many new features have been added to the program since its inception in 1986. These major improvements enable health professionals to gain a more user friendly access to the biomedical literature of the National Library of Medicine than ever before. The software enhancements aid the end-user in performing searches that will retrieve material with greater specificity and accuracy. Grateful Med provides a time-saving, cost-effective means of equal access to the medical literature regardless of the user's physical location or lack of access to other resources.

A PATIENT at clinic presents with a perplexing problem. After all the patients have been seen there is time to investigate a differential diagnosis. How can the physician find the case report or review that is needed among the subscription journals? Manually searching will be time consuming and using Index Medicus may be fruitful, although tedious.

With a personal computer, modem, phone line, and Grateful Med (GM) software, the information can be found quickly and cost effectively. Six years have passed since the National Library of Medicine unveiled the end-user computer search interface Grateful Med, version 1.0. Grateful Med has undergone a series of metamorphoses (including the arrival of a Macintosh version in early 1991), the most recent of which is Grateful Med version 6.0. Grateful Med's earlier incarnations seem primitive in retrospect. Appreciation of the additional features and the degree of upgrading of the newest release comes only through use.

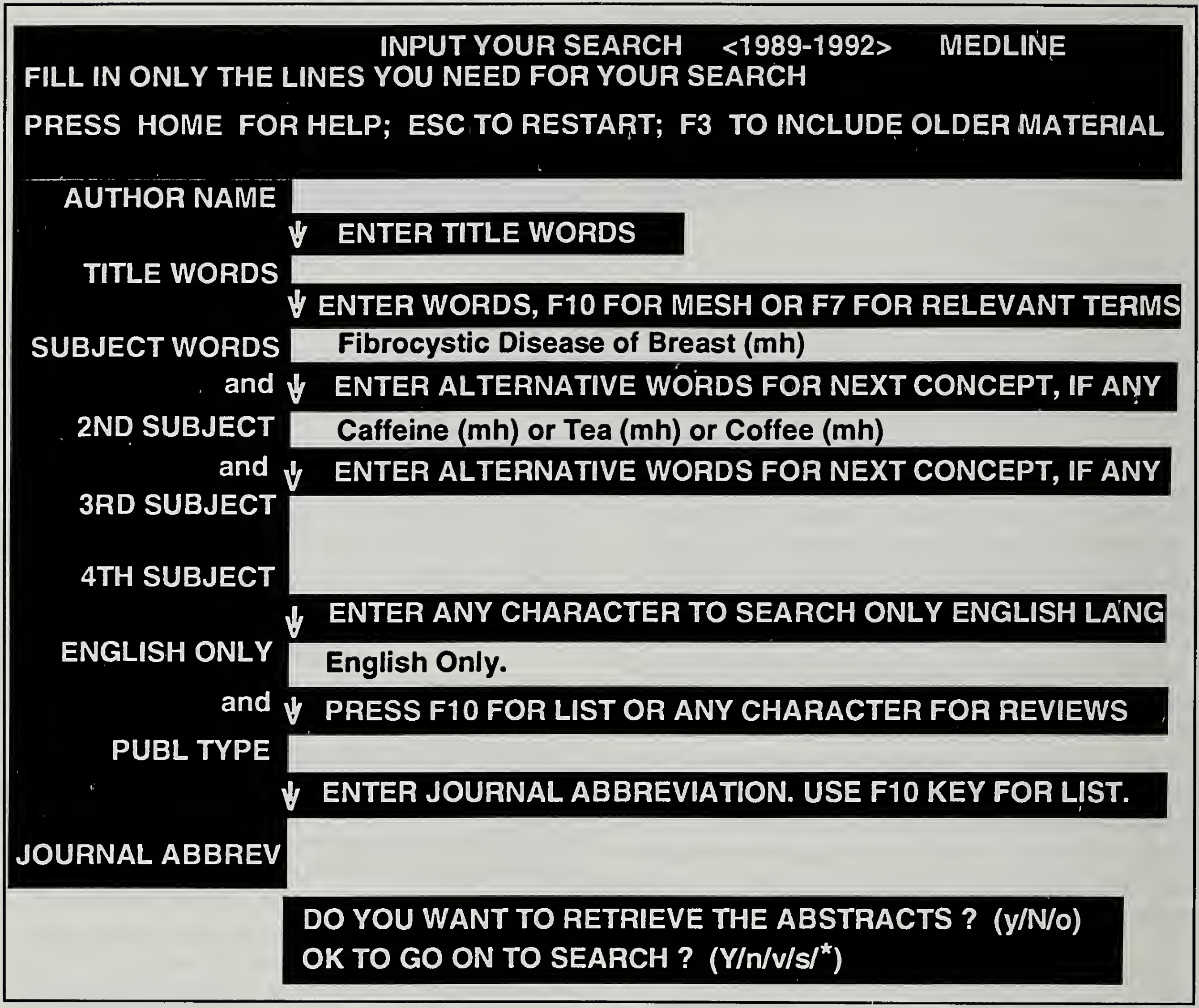
Ease of Use

Grateful Med's interface suits both novice searchers and experienced computer users. GM can be used to retrieve specific articles (eg, citation verification), to compile a bibliography by searching an author's name, or to perform a comprehensive search on the association between two or more subjects (eg, the epidemiological relationship between rabies and raccoons). These search strategies are entered via the Grateful Med INPUT SCREEN (Fig. 1). The end-user may enter author's names, text-words words from titles or abstracts), or Medical Subject Headings (MeSH-words or phrases chosen from the controlled vocabulary of terms used to index the content of articles). MeSH headings are provided by Grateful Med in a single keystroke by pressing the F10 key. The program assists the searcher in combining concepts using the *Boolean* or *logical* operators "AND" or "OR." All work entered on the Input Screen is done *locally* or *off-line* at no cost. When the searcher has finished entering terms, Grateful Med goes *on-line.* A local phone number connects with the Elhill computer at the National Library of Medicine; the user's password is entered; the search is conducted and citations (with abstracts if desired) are downloaded to the searcher's computer. Finally, GM disconnects from the NLM computer, reveals the cost, and either displays the references which were retrieved or allows the user to scrutinize the search strategy for any oversights which may have caused inadequate or unexpected retrieval. If the searcher is dissatisfied with the results of the search, Grateful Med will allow *local* editing of the search. Then the searcher may return on-line. Grateful Med will indicate which steps probably require modification, and may even suggest possible

---

FAITH MCGRATH, M.L.S., Education Coordinator, National Network of Libraries of Medicine–New England, University of Connecticut Health Center. NICHOLAS G. TOMAIUOLO, M.L.S., Information Services Librarian, Lyman Maynard Stowe Library, University of Connecticut Health Center.

Reprint requests to: Nicholas G. Tomaiuolo, M.L.S, Lyman Maynard Stowe Library, University of Connecticut Health Center, P.O. Box 4003, Farmington, CT 06034.



```
                         INPUT YOUR SEARCH     <1989-1992>     MEDLINE
 FILL IN ONLY THE LINES YOU NEED FOR YOUR SEARCH
 PRESS  HOME  FOR HELP;  ESC TO RESTART;  F3  TO INCLUDE OLDER MATERIAL

     AUTHOR NAME
                   ↓  ENTER TITLE WORDS
     TITLE WORDS
                   ↓ ENTER WORDS, F10 FOR MESH OR F7 FOR RELEVANT TERMS
   SUBJECT WORDS     Fibrocystic Disease of Breast (mh)
             and   ↓  ENTER ALTERNATIVE WORDS FOR NEXT CONCEPT, IF ANY
     2ND SUBJECT     Caffeine (mh) or Tea (mh) or Coffee (mh)
             and   ↓  ENTER ALTERNATIVE WORDS FOR NEXT CONCEPT, IF ANY
     3RD SUBJECT

     4TH SUBJECT
                   ↓  ENTER ANY CHARACTER TO SEARCH ONLY ENGLISH LANG
    ENGLISH ONLY      English Only.
             and   ↓  PRESS F10 FOR LIST OR ANY CHARACTER FOR REVIEWS
       PUBL TYPE
                   ↓  ENTER JOURNAL ABBREVIATION. USE F10 KEY FOR LIST.

  JOURNAL ABBREV

                   DO YOU WANT TO RETRIEVE THE ABSTRACTS ?  (y/N/o)
                   OK TO GO ON TO SEARCH ?  (Y/n/v/s/*)
```

Figure 1
Grateful Med 6.0 Input Screen illustrating basic search formating.

search terms. These suggestions are called *relevancy judgments* and are based on the commonalty of subject headings found in retrieved articles.

MEDLINE

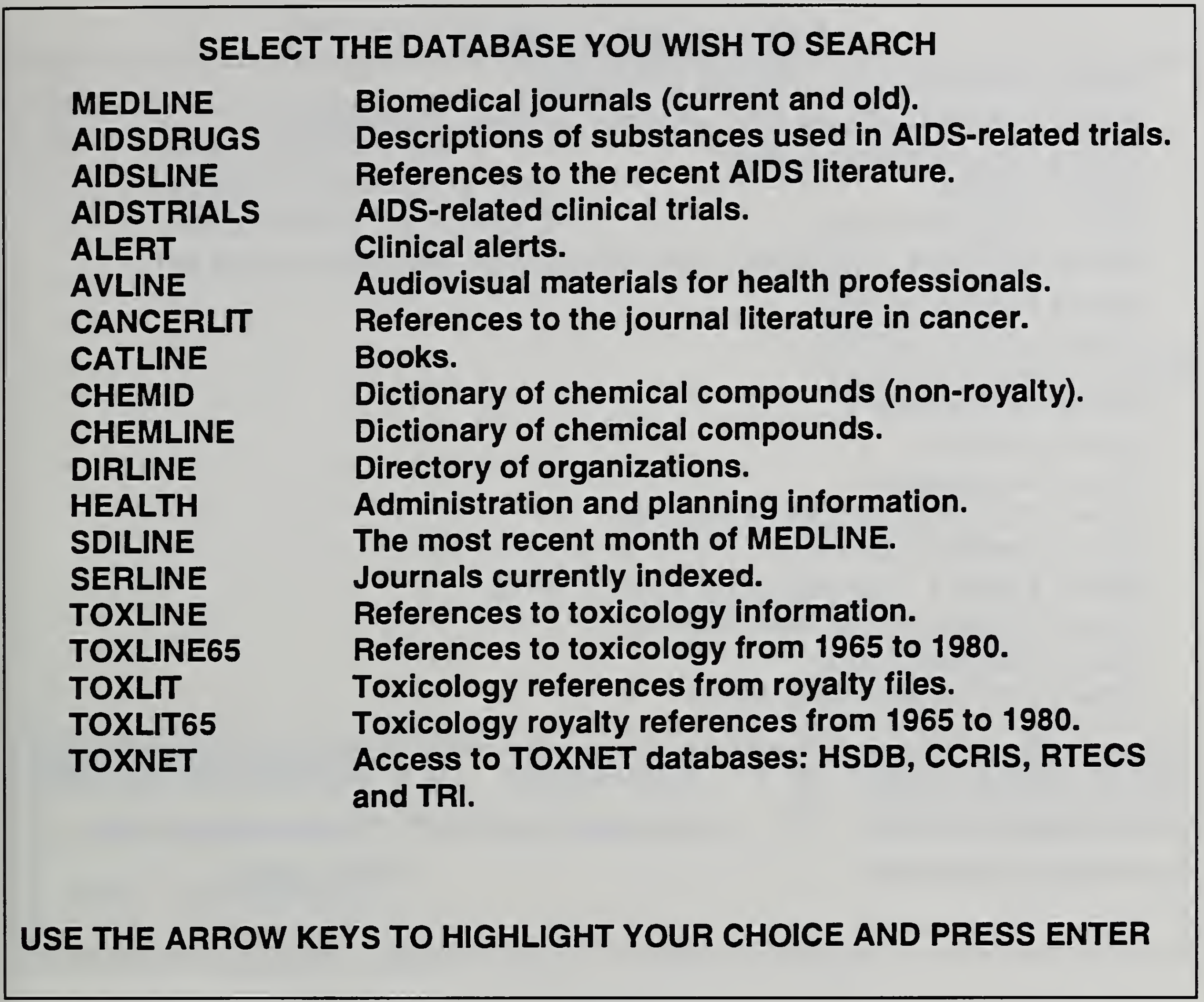
MEDLINE is the preeminent database of the MEDLARS [MEDical Literature Analysis and Retrieval System] family of databases. MEDLINE contains over seven million bibliographic citations to the biomedical journal literature. The database is the computerized counterpart to *Index Medicus,* the *International Nursing Index,* and the *Index to Dental Literature.* There are weekly updates with about 30,000 citations added each month. Major clinical journals are indexed in approximately two to five weeks, but other more obscure journals may take longer. Bibliographic citations from 1966 to the present are covered and 70 languages are represented with about 75% in English. About 60-75% of the current file includes abstracts.

Version 6.0

Grateful Med version 6.0 for DOS (IBM PC) was released in late May 1992. The following is a summary of the new features of this software.

Databases.—Grateful Med users have always been able to search from a number of other databases in addition to MEDLINE (Fig. 2). These choices have increased with six newly accessible databases in Grateful Med 6.0:

- AIDSDRUGS is a factual database covering AIDS related therapeutics being used in clinical trials—English only.
- AIDSTRIALS is a factual and referral database covering trials of substances being used against AIDS, ARC, and HIV infection.
- ChemID is a factual dictionary of chemicals.
- ALERTS contains the findings from selected NIH-funded clinical trials which are viewed to be too impor-



SELECT THE DATABASE YOU WISH TO SEARCH

MEDLINE	Biomedical journals (current and old).
AIDSDRUGS	Descriptions of substances used in AIDS-related trials.
AIDSLINE	References to the recent AIDS literature.
AIDSTRIALS	AIDS-related clinical trials.
ALERT	Clinical alerts.
AVLINE	Audiovisual materials for health professionals.
CANCERLIT	References to the journal literature in cancer.
CATLINE	Books.
CHEMID	Dictionary of chemical compounds (non-royalty).
CHEMLINE	Dictionary of chemical compounds.
DIRLINE	Directory of organizations.
HEALTH	Administration and planning information.
SDILINE	The most recent month of MEDLINE.
SERLINE	Journals currently indexed.
TOXLINE	References to toxicology information.
TOXLINE65	References to toxicology from 1965 to 1980.
TOXLIT	Toxicology references from royalty files.
TOXLIT65	Toxicology royalty references from 1965 to 1980.
TOXNET	Access to TOXNET databases: HSDB, CCRIS, RTECS and TRI.

USE THE ARROW KEYS TO HIGHLIGHT YOUR CHOICE AND PRESS ENTER

Figure 2
Grateful Med 6.0 Database Selection Screen.

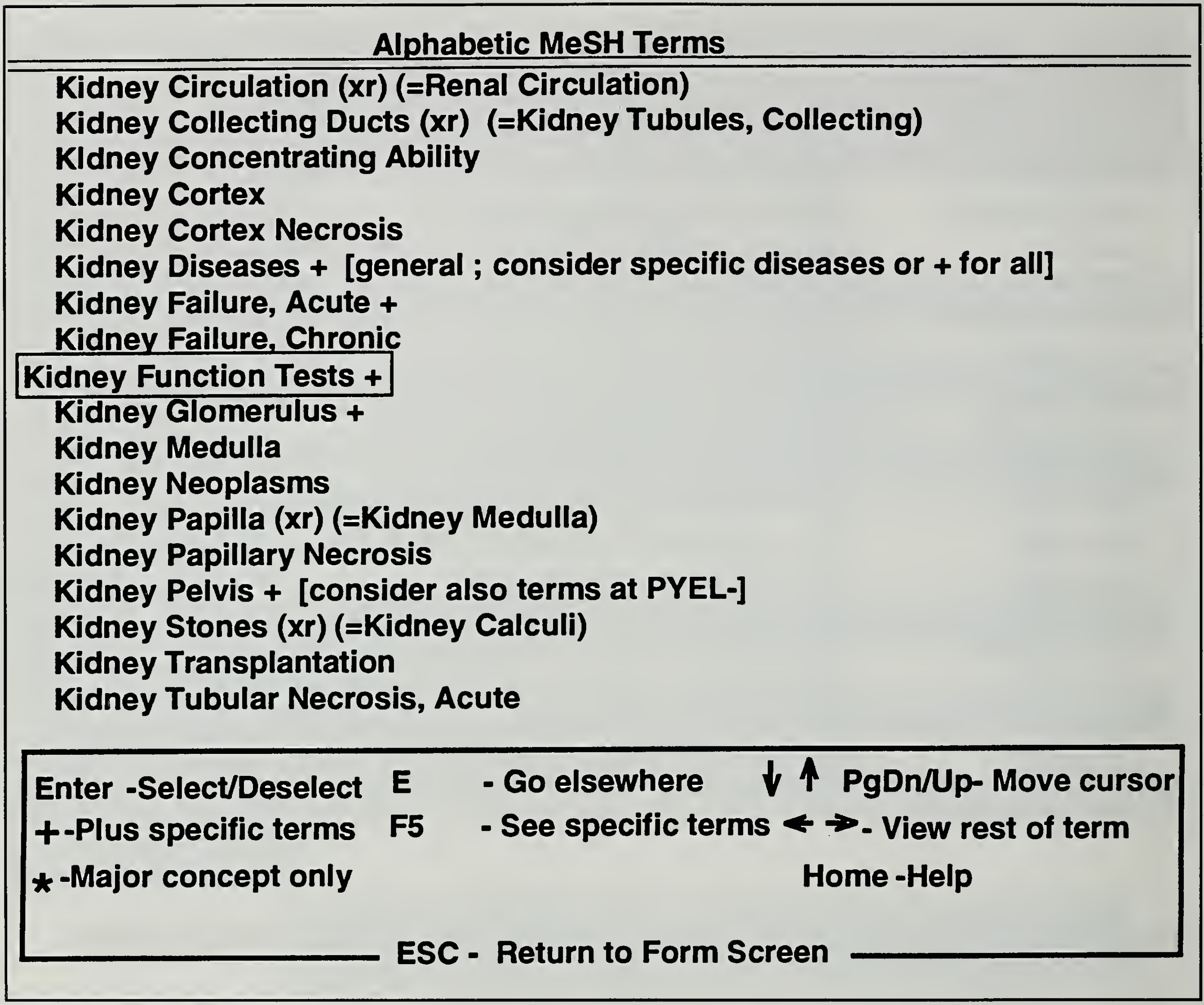
tant to the medical community to wait for publication. Grateful Med will notify you of a new Clinical Alert after the completion of any search.

- SERLINE contains citations to serials published from 1965 to the present.

- TOXNET is a collection of files on toxicology, hazardous chemicals, and related areas. The files currently available via Grateful Med are: CCRIS (Chemical Carcinogenisis Research Information), HSDB (Hazardous Substance Data Bank), RTECS (Registry of Toxic Effects of Chemical Substances), and TRI (Toxic Chemical Release Inventory).

MeSH.—GM 6.0 includes several helpful MeSH enhancements:

- Suppose the searcher is looking for information about tests for kidney function. The user types just one word, *kidney,* on subject line of the INPUT SCREEN and press the F10 key. This will display a listing of MeSH headings (the indexed or descriptor terms used to describe the journal articles referenced) in the alphabetical area of the term entered. The MeSH heading *Kidney Function Tests* is found and has a "+" sign after it (Fig. 3). This means that the heading is a general term and has more specific terms indented beneath it. With version 6.0 you simply press the F5 key and the more specific terms are displayed (Fig. 4). Under *Kidney Function Test* the terms *Blood Urea Nitrogen, Glomerular Filtration Rate,* and *Radioisotope Renography* are displayed. These options are based on the MeSH Tree Structures, which is a hierarchical tool for locating the most specific headings. There is the option of choosing a more precise term to obtain the most specific retrieval or choosing the "+" sign which "explodes" the term and will include the general as well as all the more specific terms.



Alphabetic MeSH Terms

Kidney Circulation (xr) (=Renal Circulation)
Kidney Collecting Ducts (xr) (=Kidney Tubules, Collecting)
Kidney Concentrating Ability
Kidney Cortex
Kidney Cortex Necrosis
Kidney Diseases + [general ; consider specific diseases or + for all]
Kidney Failure, Acute +
Kidney Failure, Chronic
Kidney Function Tests +
Kidney Glomerulus +
Kidney Medulla
Kidney Neoplasms
Kidney Papilla (xr) (=Kidney Medulla)
Kidney Papillary Necrosis
Kidney Pelvis + [consider also terms at PYEL-]
Kidney Stones (xr) (=Kidney Calculi)
Kidney Transplantation
Kidney Tubular Necrosis, Acute

Enter -Select/Deselect
+-Plus specific terms
*-Major concept only
E - Go elsewhere
F5 - See specific terms
↓ ↑ PgDn/Up- Move cursor
← →- View rest of term
Home -Help
ESC - Return to Form Screen

Figure 3
Grateul Med 6.0 Alphabetic MeSH Terms Screen.

- The "*" (which makes your term the major concept of the article) and the "+" are usable either before or after the selection of MeSH.

- The MeSH screen now allows for unrestricted scrolling of the terms and notes change with a high intensity display as you cursor over terms. The new version allows for easier scrolling with the up and down keys in addition to the PgUp and PgDn.

- Additional explanatory notes have been added to MeSH terms. When looking at the MeSH term *Infant* there is a note informing you that the term covers the one-month to two-year age group; for birth to one-month use *Infant, Newborn.*

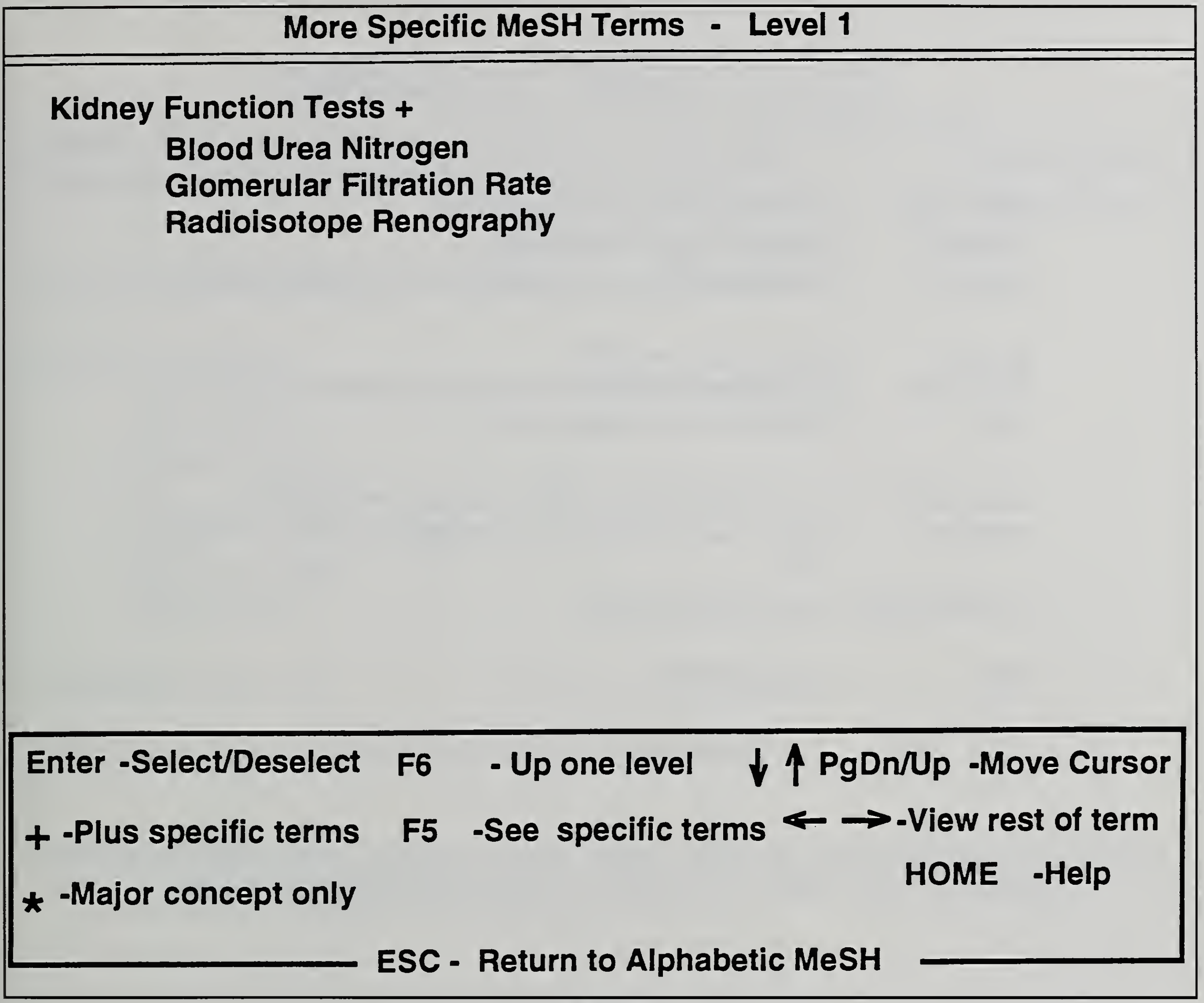
- The (PX) preexplode is no longer used; the software automatically uses it when available without any user prompting.

Input Screen Features.—

- MEDLINE backfiles no longer need to be searched separately. Backfile searching can be performed in conjunction with the current file.

- The choice of limiting to a specific publication type has been expanded well beyond the limitation of *Review.* At the *Publication Type* input line, press F10 and there is a choice of five different types of review articles [in addition to the general review]; two types of clinical trials; and practice guidelines or administrative and procedural guidelines.

- At the *Journal Abbreviation* input line, pressing the F10 key will bring up about 100 *Abridged Index Medicus* or AIM titles (the core list of medical journals) which can be selected in a similar manner to MeSH terms or *Publication Type.*



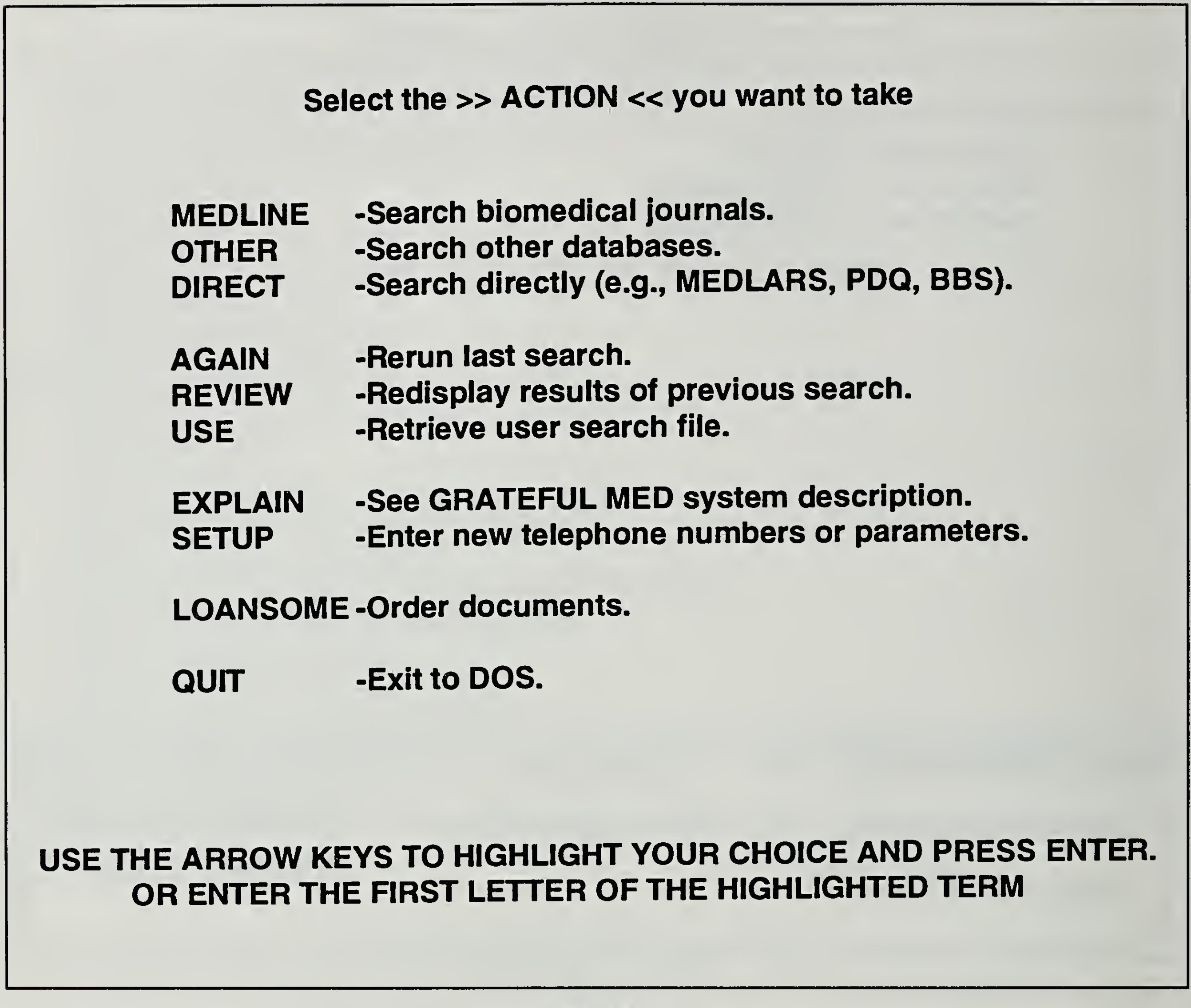
More Specific MeSH Terms - Level 1

Kidney Function Tests +
Blood Urea Nitrogen
Glomerular Filtration Rate
Radioisotope Renography

Figure 4
Grateful Med 6.0 More Specific MeSH Terms, Level One Screen.

Additional Search Features.—

- The section of the software in which you enter phone numbers, password, etc., (SETUP) was only accessible from the DOS prompt, but now it is easily available directly from the ACTION screen (Fig. 5). There are two new communications access methods to connect to the NLM computer: Internet (TCP/IP) or Novell Networks (NASI). A *Custom* option is also available which provides a place for future methods as they become available.
- Relevancy judgments can be made for specific retrieved references which are the most appropriate to your search. GM analyzes the MeSH terms in these citations and if there is some commonalty, those terms will be identified. The terms may then be used to redo and/or refine your search.
- If there is a new Clinical Alert, GM 6.0 will inform you. Immediately following your search there will be a message at the bottom of the *Your Search Found* screen. The *Next Step* menu gives you the opportunity to return on-line to retrieve the data. ALERTS is the database where Clinical Alerts are stored and are made accessible for searching.
- GM 6.0 allows search results to be saved to a file that can be used again within GM (the file must be saved in the MEDLARS or unreformatted form in order to take advantage of this feature), not just in another application, as was the previous limitation.
- The Loansome Doc (LD) feature, which is available in all areas of the United States as of December 1991, can be used to order full text articles from a medical library in your area (individuals must make an agreement with an area library) (Fig. 6). If there are references being considered for ordering from LD they can be selected by typing "O" while reviewing search results. This places them in the Orders file, but the final ordering command is presented at the LD Action Screen.



```
Select the >> ACTION << you want to take

MEDLINE   -Search biomedical journals.
OTHER     -Search other databases.
DIRECT    -Search directly (e.g., MEDLARS, PDQ, BBS).

AGAIN     -Rerun last search.
REVIEW    -Redisplay results of previous search.
USE       -Retrieve user search file.

EXPLAIN   -See GRATEFUL MED system description.
SETUP     -Enter new telephone numbers or parameters.

LOANSOME -Order documents.

QUIT      -Exit to DOS.

USE THE ARROW KEYS TO HIGHLIGHT YOUR CHOICE AND PRESS ENTER.
OR ENTER THE FIRST LETTER OF THE HIGHLIGHTED TERM
```

Figure 5
Grateful Med 6.0 Action Screen.

- The GM Bulletin Board System (BBS) is available for comments and/or questions to NLM, to receive messages, and download files. You may now use the BBS to obtain a user ID code and password, and to purchase GM software.

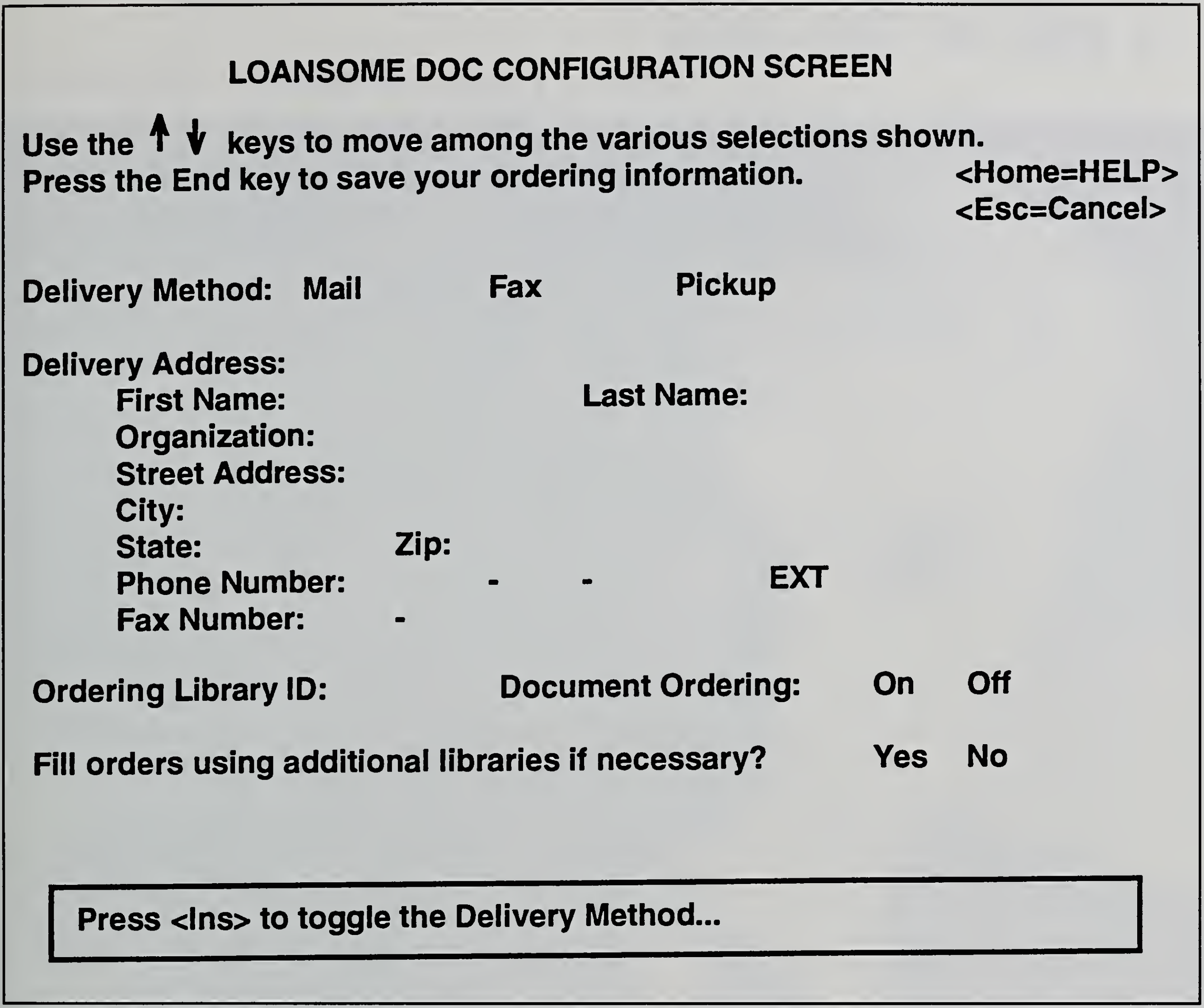
Hardware.—Some hardware requirements have changed for version 6.0.

- Your PC must have a hard disk with 1.9 to 2.4 megabytes of free space and 512 kilobytes of available RAM.
- GM is also available for the Macintosh and a major update of the Mac software will be released in early 1993. This version will include similar features to those now available in GM Version 6.0 for DOS.
- Use with a UNIX system is only possible at the present time if DOS is loaded on a subdirectory. Hopefully, a UNIX version will be coming in the near future.

Cost.—

- The GM communications/search facilitation program was initially designed as an inexpensive, flexible alternative to manual searching. Pricing of either DOS or Macintosh versions is $29.95 with $20 of free search time with a new user ID and password. Depending on how much searching is done, GM should be much less expensive for an individual or a small practice than purchasing a compact disc drive and a MEDLINE CD-ROM subscription. When the end-user searches Grateful Med, charges are assessed only for the references that are downloaded. Unlike several other on-line vendors, a minimum monthly charge is not imposed.

- As of January 1993, the charges for on-line time have dropped to $18/hour with the cost of an average MEDLINE search being reduced to an estimated $1.25. This is approximately a 40% decrease in comparison to previous



LOANSOME DOC CONFIGURATION SCREEN

Use the ↑ ↓ keys to move among the various selections shown.
Press the End key to save your ordering information. <Home=HELP>
<Esc=Cancel>

Delivery Method: Mail Fax Pickup

Delivery Address:
First Name: Last Name:
Organization:
Street Address:
City:
State: Zip:
Phone Number: - - EXT
Fax Number: -

Ordering Library ID: Document Ordering: On Off

Fill orders using additional libraries if necessary? Yes No

Press <Ins> to toggle the Delivery Method...

Figure 6
Loansome Doc Configuration Screen.

charges. The Prime Time price differential has been eliminated.

- Under a special experimental arrangement with the American College of Physicians (ACP), the National Library of Medicine will provide individual members of the ACP with unlimited access to MEDLINE and the other MEDLARS databases for an annual flat fee of $200. The agreement is in effect from April 1992 through April 1994. To take advantage of this offer, call NLM at 800-638-8480 or the National Network of Libraries of Medicine (NN/LM) Regional Office at 800-338-7657. A sign-up form will be faxed or mailed.

Support.—Help for GM users is provided via a number of different mechanisms.

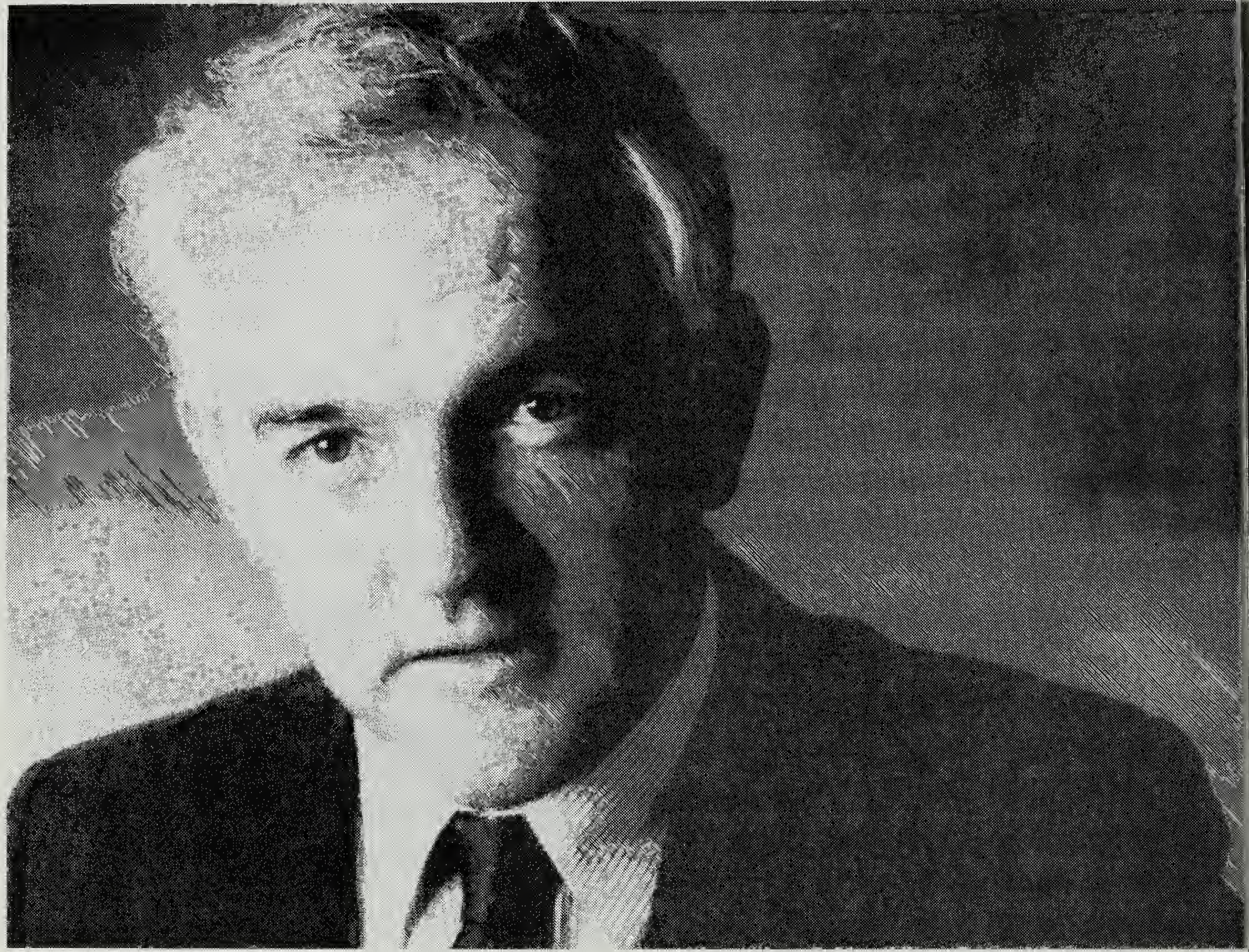
- There is an GM on-line bulletin board system (1-800-525-5756);
- the MEDLARS service desk (1-800-638-8480);
- via the Internet you can send email (gmhelp@gmedserv.nlm.nih.gov);
- the NN/LM Regional Office (1-800-338-7657);
- and there is an Internet listserv group (Listserv@ndsuvml).

Conclusion

Although end-user searching with Grateful Med 6.0 may not be the answer to everyone's biomedical information needs, the software offers the power of equal access to the medical literature regardless of the health professional's physical location or lack of other resources.

"Filling out insurance forms gives my patients a pain in the neck."

—Dr. Patrick J. Carolan, Orthopedics, Bridgeport, CT



"Good medical care was once as easy as seeing your doctor. Complications set in. Forms to fill out. Deductibles. Hidden costs. Partial coverage. Non-medical intrusions into the patient-physician relationship.

"Doctors decided to do something about it. Through our State Medical Society, we first created **CSMS-IPA,** Connecticut's only *statewide* IPA. Then we launched **M.D. Health Plan,** based on a simple principle: The best medicine for *patients* is practiced by *physicians*— not insurance companies, not benefit managers, not cost analysts.

"It worked! Now the fastest growing health plan in Connecticut and the choice of over 68,000 patients, our company is 100% owned and 100% directed by practicing physicians. Why not?"

Patrick Carolan, M.D., President, CSMS-IPA

If *you* wish to join the 3,000 CSMS physicians who now participate, call us at one of the numbers below.

M.D. HEALTH PLAN

6 DEVINE STREET • NORTH HAVEN, CT 06473 • (203) 776-5759 • 1-800-345-9272

50 Years Ago

From *The Connecticut State Medical Journal*

March 1943

Social Functions of the Modern Hospital

G.M. MACKENZIE, M.D., Cooperstown, N.Y.

The Author. *Physician-Chief, Mary Imogene Bassett Hospital, Cooperstown, New York.*

An address, here somewhat condensed, delivered September 15, 1942, at a meeting of the Westchester County Medical Society and printed in the December issue of the Westchester Medical bulletin. Reprinted with permission of the Editors of that Publication.

AS a result of the adoption by medicine of many of the accurate quantitative methods of the natural sciences, physicians have been provided within the last 50 years with an astonishing collection of new tools. It is difficult for us in 1943 to visualize the practice of medicine without clinical bacteriology or clinical chemistry, without the complement fixation test for syphilis, arsphenamine, x-rays or radium, without transfusions, without insulin, without liver extract. All of these are tools which we use every day and take for granted, but they were all acquired very recently. And now the knowledge of vitamins, hormones, metabolism, nutrition, the physiology of electrolytes and of many other mechanisms and physiologically imat the same time exciting, and, because of the impossibility of keeping fully informed, almost discouraging. This list is impressive but it does not include the therapeutic agents very recently discovered which from the point of view of human welfare are perhaps the most important of all:—the sulfonamides; and as the tempo of progress in medicine increases the task of becoming familiar with even the most important of the new diagnostic procedures and therapeutic agents steadily becomes more difficult. We are beginning to reap the harvest from the seed which was sown when medicine began to strive to attain the status of a science.

New opportunities are presented to, and new obligations are imposed upon, hospitals by the profound disruption of our usual way of life which the war has caused. Many hospitals and many communities already are acutely aware of the shortage of physicians. Before long many more hospitals will doubtless share the same problem. Two-thirds of the 80,000 medical men in this country under 45 years-of-age, we are told, must be taken by the armed forces. Here is a new challenge to hospitals. What contribution will they make in this crisis? Already they are contributing in many ways: releasing doctors and nurses, carrying on with smaller staffs, training and making greater use of nurse's aides, organizing emergency medical field units and other emergency services. There will be great need for hospital administrators and hospital staffs to study methods to increase efficiency, to centralize medical care in hospitals, to devise ways and means for physicians remaining in civilian practice to organize their work on a better time-saving basis. By extending its activities in the field of prevention the hospital can make an important contribution to the war effort; and prevention, of course, includes early recognition and early treatment. A program of prevention or health conservation, or, better, health improvement of this sort would take the hospital increasingly into educational activities. Any reduction in the incidence of disease among the civilian population including workers in war industries, will be a significant contribution to the war effort.

The progress of medicine and the growth of social consciousness have created a great public demand for hospitals and hospitalization; as this demand has increased a great variety of hospitals has developed. We have the great medical centers and richly endowed university hospitals equipped and staffed for teaching, research and the best type of medical care. They are magnificent temples of medicine; but we have also hundreds of small, poorly equipped, incompetently staffed proprietary hospitals. Between the two there are many varieties—large and small, good and bad, voluntary, proprietary, municipal, county, state and federal. We have open staffs, closed staffs and courtesy staffs. It does not seem

---

Reprinted from *The Connecticut State Medical Journal*, March 1943.

unfair to say that except for a few teaching institutions a large majority of the 4,494 voluntary, church related and proprietary hospitals in the United States are operated primarily as adjuncts of the private practice of the local physicians and only secondarily if at all as institutions dedicated to community service. In spite of the 6,358 hospitals in the United States (of which only 2,307 are approved by the American College of Surgeons) the larger proportion of the care of the sick is done by doctors seeing patients in their offices and visiting them in their homes. Clearly, any adequate reorganization of medical care must take this into consideration. The hospital fully aware of its social responsibilities will seek some way of improving the conditions of work, not only of those on the hospital staff but of all the physicians of the area served by the hospital.

Prominent among the ways in which the social responsibilities of the hospital can be met is the educational program of the hospital. A hospital providing an adequate library and well organized conferences and willing to spend money for these things will be rewarded by applications of more and better interns and by better care of patients.

A hospital problem with important social implications is that of the closed staff. What is to be done with the large number of physicians with no hospital connections? This problem needs careful study and it needs a solution which will provide educational opportunities at the hospital for all the physicians of the area served by the hospital. The courtesy staff is no solution; the motivations behind it are mostly budgetary. In any reorganization of medical practice which may be necessary because of war needs and war shortages, or inevitable because of the pressure of public opinion a socially sound solution of this problem must he sought. The hospital cannot serve all the people unless it serves all the physicians.

As the modern medical center has gradually evolved from the almshouse of 100 years ago the intimacy of the relationship of the hospital to the practice of medicine has progressively increased. Adequate medical care has become more and more dependent upon hospital facilities and hence the great increase in diagnostic and therapeutic procedures has underscored certain defects in the present organization of the practice of medicine. Can we find anywhere genuine satisfaction with the existing conditions of practice? The causes of dissatisfaction are multiple but I am sure that the time factor is one of the most important because with the increased number of methods for obtaining evidence about disease it takes far more time than formerly to collect the evidence; and so, often the physician in order to make a decent income is obliged to undertake more work than he can efficiently deal with. Efforts to devise solutions for the unsatisfactory features and shortcomings of medical practice have led to the trial of various forms of group practice; and many capable physicians dissatisfied with existing conditions and perceiving no solution in sight have migrated from private practice to salaried positions. The defects in the existing system are a challenge to hospitals to make a contribution to the organizational problem of medical practice. Any attempt to analyze this complicated problem must lead to the study of two features of medical practice which directly or indirectly seem to be determinants of the time problem and to account for much of the feeling of dissatisfaction. I refer to what is called free choice of physician and to fee-for-service. About these two features of medical practice there has been a vast amount of talk and argument and conflict. It would seem to be worthwhile to examine some of the organizations in which these two features have been modified or eliminated.

In the organization and mode of operation of the Mary Imogene Bassett Hospital in Cooperstown, New York, free choice has been greatly restricted and fee-for-service, so far as the physicians are concerned, completely done away with. Patients admitted to the hospital are, as it were, patients of the hospital; usually they do not know which member of the staff will he responsible for them. There is virtually no such thing as a private patient at the Bassett Hospital. If a patient has known a physician of the staff he may ask to have that physician take care of him. If it happens that his age and the disease which he has are right he may have the physician of his choice but if not he will be placed on the service which is organized to take care of the condition which he has. The pediatrician does all the pediatrics, the obstetrician all the obstetrics, the surgeons all the surgery and so for the other services. Patients in the Bassett Hospital do not seem to object to this. They come to the hospital for medical care, not, as a rule, to be patients of any particular doctor. In many of our University Clinics the same limitation of free choice prevails without any apparent lowering of standards. Actually the reverse would seem to be the case. Is it not true that most of the protests against limitation of free choice have come from physicians, not from patients or the public? The Bassett Hospital has experimented with a prepayment plan embracing both hospitalization and professional services. This inclusive type of plan is far easier to organize and operate with a salaried staff than when each staff member is practicing as an independent individual. The plan has seemed to work satisfactorily from the point of view both of the hospital and of the members. At the Bassett Hospital physicians hardly enter into the business relationship with patients. The administrative office of the hospital renders all the bills, makes the collections and keeps the books. I am quite sure that all members of the staff consider this a release from an unpleasant feature of the practice of medicine.

Another principle governing the professional activities of the Bassett Hospital and also of course of most teaching hospitals, is that each man's work in the care of patients is exposed to the critical scrutiny of his associates. Interns, residents and other members of the attending staff have ample opportunity to see how carefully and intelligently each member of the staff does his clinical work. There is no such thing as diagnosis and therapy undisclosed to other members of the staff. I think I can say without reservation that hardly a patient in 15 years has expressed opposition to or dissatisfaction with a relationship which is less private than that which commonly exists between a physician and his private patient. Needless to say this less exclusive and less private relationship between patient and physician does not preclude the existence of friendliness, sympathy and an understanding intimacy with the patient. The experience in Cooperstown and in many other clinics seems to indicate that some restriction of free choice does not impair the human and personal relationship of medical practice.

Those responsible for the financial operation of the Bassett Hospital have shown wisdom I believe in making provision for clinical investigation and research in the medical sciences. The amounts allowed for research are not large compared to other items in the budget, but unquestionably investigative work adds something of value to the professional atmosphere of the hospital. There is no doubt that our teaching hospitals owe much of their prestige to the intellectual climate created by investigative work. Without it a critical and discriminating attitude toward both the old and the new in medicine is apt to be largely absent. Clearly most busy practitioners working on a 24 hour day and a seven day week schedule have no time or energy for planning or executing research projects.

One of the major objectives of any refashioning of medical practice should be a revision of the general practitioner's schedule. Three traditions which became established long before the development of modern medicine are responsible for the enslaving schedule of the general practitioner: (1) The tradition of extreme professional individualism. This is the chief determinant of the existing system of medical practice; coordinated practice, collective action, team work, whatever one chooses to call it, give to the physician a far larger measure of control over his time than the general practitioner now enjoys. (2) The tradition of free choice. If the patient is entirely free to choose he may, and often does, elect to exercise his prerogative during the night or on Sunday, irrespective of the urgencies of the situation. With a coordinated system of medical practice operating from a hospital the patient's choice would be somewhat restricted. As a matter of fact, of course, in thousands of small communities the patient's freedom of choice is limited to a single physician, but this type of restricted choice doesn't help the physician's schedule. Isn't it true that the patient's freedom of choice often destroys or impairs the physician's freedom to achieve professional growth? (3) The tradition of fee-for-service. This old tradition must obviously accept a large share of the responsibility for the time problem in medical practice. For most practitioners it is too costly to be out of reach when patients want to see them. We hear it stated that working on a salary basis makes of the doctor a hired man, results in regimentation and loss of individualism. On this question I suggest that the unyielding advocates of fee-for-service consult the men in full-time positions in university clinics, or the salaried men in the United States Public Health Service, or those on the staffs of the New York State Tuberculosis Hospitals, the Mayo Clinic, many group practice organizations, or even the staff of the small rural hospital at Cooperstown. Opposition to the institutional fixed salary for practitioners of medicine comes almost exclusively from those working on a fee-for-service basis. Let us be sure that in this matter we are not being led by false prophets. In regard to all three of these traditions let us be sure that we are not handicapping ourselves by slavish adherence to ancient ideas. Let us examine them carefully and see if they are fetishes.

Before I close with an attempt, in the interest of clarification, to outline the organization and the range of action of the socially integrated hospital as I would blueprint it, I should like to emphasize two principles which seem to be of basic importance in any attempt to refashion medical practice. The first is that every hospital should so organize its resources that the benefits of medical progress, including prevention, health improvement and early diagnosis, will promptly be made available to all the people. To shorten the lag between discovery and the full application of the contributions of science, to narrow the gap between actualities and possibilities in medical practice, should be major objectives. We cannot be proud of the fact that 30 years after the Wassermann reaction and arsphenamine became available five to 10% of the people of this country are infected with syphilis; that there are still in the United States 91,000 deaths of children under 15, nearly all from preventable disease; that there are about 12,000 maternal deaths annually in the United States and that the Obstetrical Advisory Committee of the New York Academy of medicine after investigating 2,041 maternal deaths reported that two-thirds of them were preventable. These are merely random samples. The second principle is that for all physicians conditions of work and remuneration should be established which provide opportunities for professional growth, a position of dignity in the community. financial security and advancement proportionate to ability and industry.

Is it visionary to think of the hospital of the future and perhaps of the not very distant future as a socially progressive health center? The evolution of medical care from almshouse to health center in a hundred years indicates impressively the growth of scientific achievement and social consciousness. Let me try to outline such a hospital health center under seven headings.

1. *Size and physical equipment.*—The hospital should have at least 100 beds. The actual size would in general vary with the density of the population. Urban hospital health centers would economically be several or many times the minimum size. Hospitals with less than 10 beds would have difficulty in providing adequate laboratory and library facilities and in economically including the necessary specialists on the staff. Every hospital should include offices for the entire staff, space and equipment for laboratories, and for all the diagnostic and therapeutic procedures of accepted value.

2. *Organization of the staff.*—The staff should consist of interns, residents and attending physicians and surgeons holding various ranks up to the chief of service. All the physicians of the area served by the hospital would hold positions on the staff and, by the same token, all the medical work of the area would be done by the staff of the hospital. Any physician who is incompetent to hold a position on the hospital staff is also incompetent to practice medicine independently. There need be no competition for patients among hospitals or among physicians; the isolation of the general practitioner should be ended.

3. *Salaried staff.*—The entire staff would be on a salary basis and all business transactions with patients would be managed by administrative officers. The salaries of the professional staff might reasonably correspond to those of the commissioned personnel of the army and navy, and similarly there would be retirement pay. The patient-physician relationship would not be marred by business transactions.

4. *Range of professional activities.*—The staff would be organized to provide general medical care or specialist services for patients at home, for ambulatory patients and for hospital patients. In rural areas there would be outposts to which members of the staff would be assigned for periods of duty of specified duration. Upon completion of such a period of outpost duty they would return to the hospital health center. It would not be difficult with a staff organization of this sort to make provision for relief, holidays, sick leave and study leave.

5. *Educational arrangements.*—The educational facilities and program for the professional staff would include an adequate library and a librarian, clinical and pathological conferences, informal teaching rounds for interns, opportunities for interns and junior members of the group to present patients or prepared papers at staff meetings, critical and thorough death analyses, lectures or clinics by guest speakers, and periods of study at other clinics or at medical schools. Clinical and laboratory work should be so organized and integrated that there would be abundant opportunities for each member of the staff to profit by contact with other members.

6. *Preventive medicine and health improvement.*—Preventive work would be organized in cooperation with local, state or federal public health officers whose offices should be in the hospital. The visiting public health nurse trained to pick up clues of disease in initial stages and bring about early diagnosis and treatment would be an important part of the organization. Systematic education of all the people in public and individual health would emanate from the hospital health center. The local health officer would be a member of the staff and also the school physician. The entire staff would be actively concerned with prevention of disease, health conservation and health improvement. The physician would be less exclusively occupied with salvage medicine.

7. *Education of the people in science.*—The hospital health center would integrate its functions further with the life of the community by lectures for lay groups not exclusively on medical science but on general science and particularly on the social relations of science. It would cooperate with the schools in the teaching of science, public health and individual health. It would he the community center for healing, for health, for science and the scientific attitude.

But who is going to pay for all this and, secondly, under what authority and control is such a hospital health center to be built and operated? An answer to the first of these questions is, I think, gradually emerging from the many surveys and discussions and more recently, from the experimental approach to this aspect of medical economics. Prepayment plans, the insurance principle, the steady progress of public approval of taxation as a source of funds for medical care for all the people point the way to the economic basis of the hospital of the future. As to what the controlling agency—whether federal, state, local or nonprofit association—of such a hospital health center would be, there is at present no definitive answer, but I am sure that the attitude of the medical profession, positive or negative, will he an important factor in determining the type of organization that hospitals of the future will have. The controlling agency might reasonably include representatives of (a) the lay public who constitute the consumers; (b) the medical profession which provides the personnel to carry on the functional activities of the hospital health centers; (c) the federal, state, or local government because of the necessity of tax support if the low income groups and indigent are included; (d) and finally the universities because they represent high quality of medical care, science, and progress in medicine. Such a hospital, it seems reasonable to believe, would silence the critics of the medical profession and provide remedies for many of the vicissitudes and shortcomings of medical practice as now organized.

Ivory Towers and Organized Medicine

> Ivory tower: an image connoting a state of withdrawal from the practical issues of life in society into a world of purely intellectual concerns.
>
> (Webster's Dictionary)

The survival of medicine as a noble profession, the freedom of doctors to practice what they learned, and the furthering of individual doctor-patient relationships all depend upon how much we are controlled by the political forces. These forces include a variety of paramedical persons wanting to do what doctors have done traditionally, people who want to practice medicine without going to medical schools, social organizations that want to control our day-to-day patient care activities in the name of cost containment, and finally the legislature who want to put restrictions in so many ways that treating patients has become a burden on so many of us. The county and the state medical societies have constantly worked hard to have our voices heard in the political process. When a legislative bill comes up, the rank and file members are alerted so that they can contact their legislators to give information and to support or oppose a particular bill. The state society with the help of our in-house legislative liaison and our lobbyists tries to have input in all the activities of our state legislature. Leaders of the county and state medical organizations routinely appear before the legislative committees to testify regarding the upcoming bills. Our achievements in this regard are many. Some of them are as follows: We have achieved some measure of tort reform. We have stopped the mandatory assignment of Medicare. We have helped draft laws in public safety and welfare. We have prevented triplicate prescription laws. We have opposed and prevented any allied health profession's attempts to go beyond their scope of practice. All these activities have helped not only the members of the state and county medical societies but everybody including the members of the Yale and University of Connecticut medical school faculties, many of whom who are not members of these societies.

W.E.B. Dubois, the American writer and teacher wrote in 1903: "The function of the university is, above all, to be the organ of that fine adjustment between real life and the growing knowledge of life." It is unfortunate that our colleagues at these fine teaching institutions are not involved enough with real life situations of organized medicine. They may feel that they are above politics and their only concern is with intellectual pursuits. At these times of apocalyptic changes in health care delivery we need them more than ever. So I extend an invitation to each and every one of our academic brothers and sisters who are not members of organized medicine to become involved and to join the Connecticut State Medical Society. I would also urge the private practicing doctors to talk to their friends in the universities about the urgent nature of this appeal. Let us make them understand what the purpose of organized medicine in our state is and how the survival of one depends on the other. "One man may hit the mark, another blunder; but heed not these distinctions. Only from the alliance of the one, working with and through the other, are great things born." Antoine de Saint-Éxupéry, French novelist (1900-44).

Sultan Ahamed
President

PS: There is a special relationship between the State of Tamil Nadu (previously the Province of Madras) in India and the State of Connecticut. Elihu Yale (1649-1721) was born in Boston and educated in England. He later served as the Governor of Madras from 1687 to 1692. The University of Madras, my alma mater, was founded in 1857. Elihu Yale as a young man went to Madras in the service of the British East India Company in 1672. He returned to London as a wealthy man. At the request of Rev. Cotton Mather, he sent the college in the Connecticut Colony goods from India and other materials which were sold for 562 pounds, a large sum at that time. In 1718 the college began using Yale's name. Both the university of Madras and the Yale University are continuing in their traditions even today in educating young physicians.

INQUIRE TODAY.

ENDORSED BY

THE CONNECTICUT STATE MEDICAL SOCIETY

—OFFERING—

- Hospital, Surgical and Major Medical Insurance
- Term Life Insurance
- Employee Benefit Package

—FEATURING—

- OFFICE OVERHEAD AND DISABILITY INSURANCE PACKAGE

  [Rates based on a 30 day waiting period for ages 30-39]

  $1500.00 weekly disability indemnity
  semi-annual premium is $528.15*

  $20,000.00 monthly overhead indemnity
  semi-annual premium is $561.00

* Optional Residual and COLA benefits available

AYR INSURANCE AGENCY

105 SANFORD STREET HAMDEN, CT 06514 288-4302

The Anencephalic as Organ Donor: Issues at the Beginning and End of Life

JOSEPH M. HEALEY, J.D.

THE case *In Re T.A.P.C.*,[1] described in a previous column, has focussed attention on the ethical and legal status of an anencephalic. Of particular interest in this case were the questions whether an anencephalic should be considered alive and entitled to protection against the premature taking of life, or should be considered dead and eligible to be used as an organ donor at the request of a parent or guardian. The Florida Supreme Court addressed these questions in a case of first impression, providing a useful framework for other states to consider.

The starting point of the court's decision was a consideration of the nature of anencephaly. Relying upon the report of a task force on anencephaly,[2] the court emphasized the uniformly lethal nature of this condition, properly defined and properly identified, with survival after the first few days of life being rare. The legal status of those surviving anencephalics became the focus of the court's review of existing Florida law. One area of inquiry involved the recognition of death in Florida's common law and statutory law. Two potential sources of clarification were identified: a common law "cardiopulmonary" definition of death and "brain death" criteria established in statute. With respect to the second, an existing Florida statute recognized irreversible cessation of the entire brain, including the brain stem as the occurrence of death, when respiratory and circulatory functions were being maintained by artificial support. However, since T.A.P.C. was not being maintained by artificial support, the brain death criteria could not be used. A further section of the statute indicated that except for a diagnosis of brain death, the standard set forth would not be the exclusive standard for determining death. However, the court found no authority recognizing any other definitions of death as part of Florida's common law.

The court went on to consider other potentially useful statutes. For example, Florida's Vital Statistics Act defines a "live birth" as

> "the complete expulsion or extraction of a product of human conception from its mother, irrespective of the duration of the pregnancy, which, after such expulsion, breathes or shows any other evidence of life such as beating of the heart, pulsation of the umbilical cord, and definite movement of the voluntary muscles, whether or not the umbilical cord has been cut or the placenta is attached."[3]

The Act further defines a "fetal death " as

> "death prior to the complete expulsion or extraction of a product of human conception from its mother if the 20th week of gestation has been reached and the death is indicated by the fact that after such expulsion or extraction the fetus does not breathe or show any other evidence of life such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles."[4]

While the court acknowledged that under these sections of the Vital Statistics Act, the delivery of T.A.C.P. involved a live birth, not a fetal death (at least for the purposes of the Vital Statistics Act), the court considered this conclusion to be "inapplicable to the issues at hand." The Act was seen to offer assistance by analogy, not clear guidance dispositive of the issues at hand. Since neither the parties nor the court's own research revealed any statute or case directly on point, the court concluded that there was no legal authority binding upon the court deciding whether an anencephalic child is alive for the purpose of organ donation.

The court then turned to public policy considerations and concluded that there was no basis to expand the common law to equate anencephaly with death. The court did hold that Florida common law recognized the cardiopulmonary definition of death. Since T.A.C.P. was breathing on her own, was not being supported by artificial support, and did not satisfy the cardiopulmonary definition of death, she was alive under Florida law and the transplantation of her organs was judged illegal. The effect of the court's decision is to treat the anencephalic who survives birth as an infant whose organs may be donated for transplantation only when legally-recognized criteria have been used to determine that its death has occurred.

REFERENCES

1. No. 79-582 (Fla. Sup. Ct., Nov. 12, 1992).
2. The Medical Task Force on Anencephaly: The infant with anencephaly. *N Engl J Med* 1990; 322:669.
3. Fla. Stat. s 382.002(10).
4. Fla. Stat. s 382.002(7).

JOSEPH M. HEALEY, J.D., Professor, Department of Community Medicineand Health Care, University of Connecticut School of Medicine, Farmington.

Correspondents: Joseph M. Healey, J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington, CT 06030-1910.

Staff Physician

Masonic Charity Foundation of Connecticut, Inc., nationally recognized as one of the finest geriatric health care providers, is currently seeking a Physician to join the staff at its 568-bed, multi-licensed, JCAH approved Wallingford campus, which includes a 150-unit continuing care retirement community which is currently undergoing expansion. Geriatric experience preferred but will consider internists and new graduates. Will work the standard workweek, with rotation of evening and weekend call.

We offer an excellent work environment, central Connecticut location, and full range of employee benefits. Salary will be commensurate with experience. **Please send curriculum vitae with salary requirements to: Dr. Erlinda Rauch, Medical Director, Masonic Charity Foundation, P.O. Box 70, Wallingford, CT 06492.** EOE. M/F/H/V

THE MASONIC
CHARITY FOUNDATION
OF CONNECTICUT

June 12, 1993
13th ANNUAL
ADVANCES IN GASTROENTEROLOGY

Bally's Park Place Hotel and Casino
Atlantic City, New Jersey

Sponsored by the
Presbyterian/Medical Center
Gastrointestinal Section
and the Underwood Memorial Hospital
Woodbury, New Jersey

Accreditation: Category 1 credit offered

Information: Registration Manager
SLACK Incorporated
6900 Grove Road
Thorofare, NJ 08086-9447
(609) 848-1000

HEALTHCARE EXPO '93 *in its 8th year*

Sponsored By CHA **The Connecticut Hospital Association and Affiliates**

Wednesday, April 21, 1993
10:00 a.m. - 5:00 p.m.

Thursday, April 22, 1993
10:00 a.m. - 4:00 p.m.

HARTFORD CIVIC CENTER

★ **Free Admission To Over 285 Exhibits** ★ **Recruitment Opportunities**

★ EDUCATIONAL SYMPOSIA sponsored by C.H.A.
★ Jeff Goldsmith PH.D. Keynote Address sponsored by ERNST & YOUNG
★ Special Programs for physicians sponsored by the Connecticut State Medical Society
★ Award Winning Seminar/Workshop presented by James W. Brown PH.D. M.H.A. "AIDS and Biosafety" OSHA's Bloodborne Disease Standard. Sponsored by the CT Chapter American College Healthcare Administrators.

CLIP AND SEND before April 10th to receive your badge in the mail (Turn in at show after this date). CLIP & FAX (or send) to: Robert Donnell Productions, 312 Park Rd., West Hartford, CT 06119 • Fax (203) 232-0982 • Phone (203) 233-3654 / (800) 243-9774

Your Name & Title: ____________________

Company & Address: ____________________ Phone: __________

REFLECTIONS ON MEDICINE

Listening to the Patient, Primary Care, and Reform

ROBERT U. MASSEY, M.D.

HEALTH care reform must include more than universal coverage, cost containment, rationalizing demands and expectations, and tort reform; structural changes, not to be accomplished in the short term, need to be undertaken by the profession itself, including its medical schools and hospitals.

Recently Dr. Stanley W. Jackson, Yale University School of Medicine, in an essay on the essential place of *listening* in psychological hearing, observed that

> ...over the centuries, it is clear that the would-be healer, in some way or other, has taken some pains to learn about the sufferer's ailments or difficulties in the process of developing a basis for his or her healing efforts.[1]

To remind us that listening was important not only in psychological healing, he recalled the adage, attributed to William Osler, "Listen to the patient; he is telling you the diagnosis." I first heard this from my professor of medicine, who told us that after eliciting the medical history we should be 90% certain of the diagnosis. A recent study suggested what some of us have long believed, that a careful history and physical examination could be slightly more reliable than data from a panoply of elegant, expensive tests.[2] My observation has been that laboratory studies, costing hundreds of dollars, are frequently ordered *before* the patient is ever seen, never mind listened to! The discerning patient should be counseled by her primary physician, if she is lucky enough to have one, to refuse! As clinical clerks 50 years ago we were reminded, "There are no routine lab tests."

The lower cost of the Canadian health care system is attributed to the relative simplicity of the government operated insurance plan; what is rarely mentioned is that 50% of Canadian doctors are primary care physicians. In spite of warnings over 25 years ago that steps should be taken to increase the number of primary physicians, the top-heavy mix of specialists in the United States[3] has continued and grown. More than half the students entering medical school say they want to practice primary care medicine; by the time they graduate, most have accepted residencies in one of the specialties, or rather, subspecialties. The problem lies not with the students; they are caught up in the culture of the medical schools and teaching hospitals. Faculties reproduce their own kind, and hospitals hardly know what to do with family practitioners or general internists.

While trying to improve care and control costs, we shall make little headway unless residency positions also are rationed. Never mind that we do not know exactly how many cardiologists are needed for 100,000 people; studies to determine this are legion, and a hundred more could be devised, all of them flawed.

Simply cut the first-year postgraduate positions (PGY-I) to the number of students graduating annually from American medical schools, and designate 50% for primary care in all years. Some additional positions might be set aside for international graduates, still maintaining half for primary care. Since most teaching hospitals are affiliated with medical schools, the country could be divided into 125 regions, and in each the numbers of PGY-I house officers would be the same as the numbers graduating from the regional medical school.

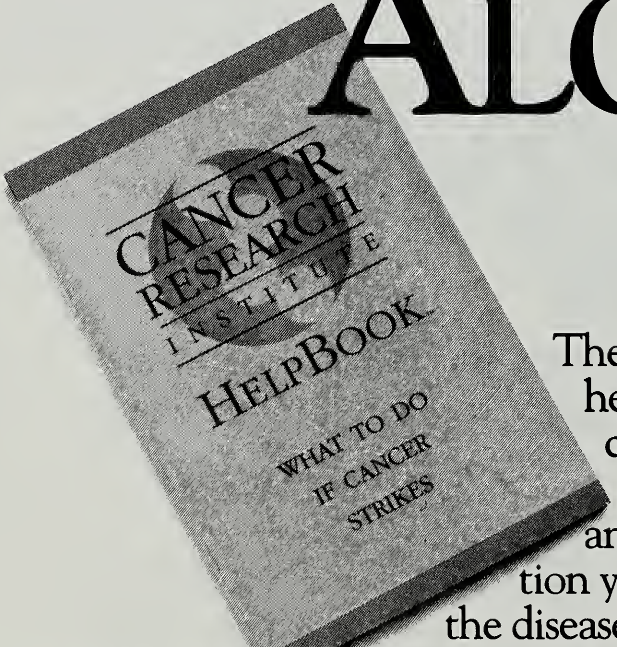
Under the Connecticut Regional Medical Program 20 years ago, the state's teaching hospitals were assigned either to the Yale or University of Connecticut region. An effort was made among the hospitals in the Capitol Area Health Consortium to limit PGY-I positions to 100 (the medical school graduating class was 88), and designate 50% for primary care. Within a few years so many exceptions had been made, and so many residents escaped into the specialties in their second year, that the effort was abandoned. Only if this were made a national program would it work today, but something like it is essential if the objectives of reform are to be met, and if medicine, the listening profession, is to recover its high ground.

REFERENCES

1. Jackson SW: The listening healer in the history of psychological healing. *Am J Psychiatry* 1992; 149:1623-32.
2. Pryor DB, Shaw L, McCants CB, et al: Value of the history and physical in identifying patients at increased risk for coronary artery disease. *Ann Int Med* 1993; 118:81-90.
3. The Citizens Commission on Graduate Medical Education: *The Graduate Education of Physicians.* Chicago, Ill., Council on Medical Education, AMA, 1966.

ROBERT U. MASSEY, M.D., Professor Emeritus, Division of Humanistic Studies, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

IF THE THOUGHT OF CANCER SCARES YOU, YOU'RE NOT ALONE.



The Cancer Research Institute is here, with help — and hope. Just write us for your free* copy of our *HelpBook*.

We've written it so millions of men and women like you can have the information you need to make decisions and deal with the disease.

If you, or perhaps someone you love, has been diagnosed with cancer, the *Cancer Research Institute HelpBook* can tell you about your choices, how to seek excellent care, and how to continue with a complete and productive life.

Even if cancer is not part of your life, you'll find it a valuable reference book to have on hand should things change.

Write today. *Cancer Research Institute HelpBook*, F.D.R. Station,
Box 5199, New York, NY 10150-5199.

CANCER
RESEARCH
INSTITUTE

** $2 per copy for postage and handling.*

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgement), provided that the are submitted in dupli–cate, signed by all authors, typewritten in double spacing, and do not exceed 1-1/2 pages of text (excluding references). They should not duplicate similar material being submitted or published elsewhere. Letters referring to a recent *Journal* article should be received within six weeks of the article's publication.

LIGHT AT THE END OF THE TUNNEL OF TIME

To the Editor: In daily life, mind is the power, and circumstance the opportunity! With imagination acting on reality, belief and conviction generate the energy of will. Faith makes the plausible possible, constructive laughter dispels depression.

For coping, postures are shaped by models in the mind's eye: Lion is the model for courage, and the Sloth for ease. Water Buffalos is the model for patience, and the Bear defiance. Human insight lets the brain order up the model to shape the attitude and mood.

Optimism is a shield against the future uncertainty, attentiveness is the engine of effort. Rest and renewal is the strategy which maintains a light at the end of the tunnel of time. Responsibility calls for endless dedication, as all try to deserve their rewards. Good luck!

Sidney Vernon, M.D.
Willimantic

FEAR OF MALPRACTICE LITIGATION

To the Editor: I get the impression from his article in the November issue that Attorney Healy underestimates the intensity of the feelings which physicians have towards claims of wrongdoing when withdrawing care from terminal patients.

The fear which physicians have in this setting is more than just a "concern [for] ... filing a suit or adverse publicity ... or the impact on one's family." The fear of defending a legal claim, for most physicians, under most circumstances, is immeasurably greater than the word "concern" can ever convey.

If physicians were merely concerned over a legal claim, they would not, as some do, resort to alcohol and drugs for relief; or even worse, suicide, when despondency becomes too great a burden.

If we are to address the fear of malpractice litigation, let's do so clearly and boldly, without confusion or timidity, shame or embarrassment; but most of all let's use language that is accurate in its description, and does not underestimate the dread, the dismay, the anxiety, the apprehension that most physicians feel when faced with even the slightest hint of a malpractice claim.

Edward J. Volpintesta, M.D.
Bethel

"Ron's Rule—I give myself one week to meet new people and start having fun on a locum tenens assignment. It hasn't failed me yet."

Ron Richmond, MD, joined the CompHealth locum tenens medical staff when he completed his residency. He wanted to travel. He loves to meet people. A little time off sounded really good. And he thinks being exposed to different types of medical practice will serve him well when he returns to his hometown to establish a community health center.

A singer. A board-certified family practitioner. A soft-spoken New Yorker. Ron Richmond knows...

It's a great way to practice medicine

CompHealth
LOCUM TENENS

1-800-453-3030
Salt Lake City ■ Atlanta ■ Grand Rapids, Mich.

LOOKING FOR REPORTS OF ANAPHYLAXIS

To the Editor: I would like any report of deaths or near deaths from anaphylaxis due to food or insect allergy. I would also like reports of deaths that were prevented by the use of epinephrine kits. Information may be sent to: Claude A. Frazier, M.D., 4C Doctors Park, Asheville, NC 28801.

Claude A. Frazier, M.D.
Asheville, NC

Medical Scholarships Available for Hartford County Residents

The Hartford County Medical Foundation, a subsidiary of the Hartford Medical Association, is accepting applications for the 1993 Medical Memorial Scholarships.

Applications are available by writing to Mrs. Michele Norbeck, Hartford County Medical Association, 100 Asylum Avenue, Suite 108, Hartford CT 06105. Deadline for completed application and accompanying materials is June 1. Scholarships are awarded in September.

This Month's Reading in Review

TIMOTHY B. NORBECK

- As the health care reform debate intensifies, more critics in Connecticut will point to health care costs in Connecticut as the third highest in the country, trailing only Massachusetts and New York.... There are some good reasons for this.... For one thing, the Medicaid eligibility standard in Connecticut is one of the highest in the nation at about 80% of the federal poverty level (nationally, that number is about 42%).... Our uninsured rate at 8% is the lowest in the country.... Generally speaking, the more affluent the state or country, the higher the amount of spending on health care—and Connecticut (despite its poverty) ranks number one in the U.S.... Three of our cities (Hartford, New Haven, and Bridgeport) have been among the poorest 10 in the country (although New Haven and Bridgeport have improved their standing recently), and poverty breeds sickness and disease.... Connecticut residents smoke more cigarettes and drink more alcohol than in three-quarters of the other states....At 13.6%, we have an older population (65 and over) than the national average (12.6%) and only 11 states have more elderly as a percentage of the total population.
- Some two years ago and despite many criticisms that he should have stayed in Canada, Quebec Premier Robert Bourassa traveled to Bethesda, Maryland, to have surgery for a malignant growth on his back.... Unfortunately Mr. Bourassa recently developed a skin cancer on his chest.... Again he had the procedure performed in Bethesda.
- Johnson & Johnson claims that their "Live for Life" health promotion program for employees has saved the company more than $5 million through reduced health care costs and reduced absenteeism during 1990 alone.... "Live for Life" costs $225 on average per employee for a total cost of $7.4 million for its 33,000 employees.... Estimated savings were about $380 per employee ($224 in health care costs and $156 in absenteeism) and a total of $12.5 million.
- Samuel Johnson (1709-1784) once remarked that "he did not care to speak ill of any man behind his back, but he believed the gentleman to be an *attorney*."
- According to a study sponsored by the Kaiser Family Foundation, the total cost for the Medicaid program will reach $120 billion for 1992, up from $51.6 billion in 1988.... The study found that it costs $1,086 per year on average to care for a poor child, $1,844 for a parent, $7,789 for a disabled person and $8,524 to pay for hospital or nursing home care for a low-income elderly patient.
- Caesarean sections are now used in 23% of all U.S. births, up from 5% in 1965.... Evidence linking caesareans to malpractice fears was scant until a recent report in the *Journal of the American Medical Association* (JAMA).... The report found that the odds of having a caesarean section were three times as great in areas where malpractice premiums (and the frequency of suit) were high compared to regions where malpractice costs were lower.
- The Kaiser report estimates that Medicaid pays for 40% of all spending to care for persons with AIDS and represents the single largest source of coverage for AIDS patients.... In 1987, Medicaid covered $390 million in costs for AIDS patients compared to $2.1 billion in 1991.
- According to the Rand Corporation, 95% of business firms with more than 250 employees offer health insurance, but only 40% of those with fewer than 10 workers do so.
- While federal Medicaid will expand to reach every poor child under age six by 1994, low-income youngsters from six to 18 will not get coverage until the end of the decade.
- According to a Northwestern National Life Insurance poll of 12 to 18-year-olds, youngsters worry about their future health.... Sixty-three percent don't expect to be as healthy as adults as they are now.... Only 10% admit use of illegal drugs, but 67% say most of their peers do.... The poll was taken at malls and also shows that 81% feel depressed, 58% "have a lot of stress," and 16% have thought of suicide!
- *Only in America:* When six-year-old Robyn Lerman of Highland Park, Illinois had two baby teeth removed by her dentist, her mother asked for them so that she could place them under her daughter's pillow for the Tooth Fairy just as her mother had done for her.... The dentist had to refuse, however, citing the OSHA guidelines established last year which classified teeth as "lab specimens" and a potential biohazard.... As such, they cannot be turned over to a patient, and a violation would result in a fine.... The disgruntled dentist complained: "The federal government can't get rid of Saddam Hussein, but here they are trying to put the Tooth Fairy out of business."

TIMOTHY B. NORBECK, Executive Director, Connecticut State Medical Society.

From the Executive Director's Office

COUNCIL MINUTES

Thursday, January 28, 1993

Attendance

Present in addition to the Chairman, Dr. Joseph C. Czarsty, were Drs. Ahamed, Beck, Bobruff, Blum, Bobruff, Brooks, Freedman, Hollister, Kamens, Keating, McDonnell, Meridy, Orphanos, Sosa, Wetstone, Wolfson, Zanker, Zeppieri.

Also present were: Mrs. J. Czarsty, Mr. Norbeck, Mrs. Lindquist, Mr. Brunell, Mr. Sullivan, Mr. Garofalo, Mr. Thompson (FCMA), Mr. Freberg (NHCMA), Mr. Coffey (HCMA).

Absent were: Drs. Cottle, Deren, Eslami, Fischbein, B. Fox, Franklin, Hoskings, Leach, Lesnik, Parke, Petro, Redmond, Sadowski, Schwartz, Tesoro, Timmerman, Van Nostrand.

Reports on Related Organizations

CPRO: Dr. Kamens submitted a written report concerning a study of the profiles of hospital mortality for Medicare patients in Connecticut (up to one year post discharge). The study has been completed for 17 diagnostic categories. He reported that the proposed plan is to feed back information to all of the hospitals and assist them in looking at the causes of variability at the individual level of performance and at the causes of variability arising from systems differences. A study is also being made about the differences in mortality associated with acute myocardial infarction in various hospitals. The eventual goal is for hospitals to conduct their own internal studies when the overall data is compiled. It was VOTED to receive the report as information.

Task Force on Donation of Services for the Medically Uninsured: Dr. Beck, CSMS representative on the Task Force, reported that the Task Force is chaired by Senator Gunther and is composed of representatives of many of the other allied health professionals, and one other physician. He reported that the Task Force has had five meetings which have been productive in preventing allied health personnel from practicing medicine without a license. Senator Gunther has submitted bills to the legislature to extend the life of the Task Force for another year and to accomplish a waiver of liability (a good samaritan law) to allow physicians to work at health care clinics without liability. It was VOTED to receive the report as information. It was further VOTED to appoint a committee to consider formulating a plan to utilize the services of retired physicians to do volunteer work and report back to the Council with recommendations.

M.D. Health Plan: Dr. Vincent Catrini and Douglas Hayward from M.D. Health Plan joined the Council to give a report. Dr. Catrini reported that he held a meeting with the leaders of the Hartford and New Haven County Medical Associations to discuss several issues. He believed the meeting was very worthwhile and a report is being prepared as to what actually was discussed and proposes to send a copy to the Chairman of the Council. A letter was distributed to Council members that was written by Mr. Hayward which addressed some of the problems that arose from their conversion to a new data processing system. The letter explained the causes for the delays in processing claims and their efforts to correct the problems. Dr. Catrini stated that he believes that they now have assurance that the conversion is completed and will now be catching up on their claims. It was reported that M.D. Health Plan has submitted a bid to cover all state employees and are awaiting a response to their bid. Mr. Hayward responded to several questions raised by the councilors. It was pointed out that several states have requested information as to how M.D. Health Plan was developed with a view as using it as a model. Dr. Catrini informed the Council that he would be available to give the Council an update on M.D. Health activities any time, each meeting, if so requested. Dr. Czarsty informed the Council that Dr. David Thompson, new president of the IPA, was going to be invited to the next Council meeting.

Report of the President

Dr. Ahamed reported that he believes that one of his goals during his presidency was to get unified county and state membership and he is hopeful that it may finally be accomplished. Meetings have been held with the Board of Directors of the Hartford and New Haven County Medical Associations and both expressed their support for unified membership. A meeting has been scheduled with the Board of the Fairfield County Medical Association to discuss the issue. Dr. Freedman, membership chairman, commended the executive directors, Mr. Norbeck, Mr. Coffey, Mr. Freberg, Mr. Schuman and Mr. Thompson for all their efforts in promoting unified membership.

Dr. Ahamed further reported on his attendance at the AMA Legislative Conference and stated that Connecticut was not alone with their problems. Many of the states are having the same problems, such as mandatory assignment, provider taxes, etc. Health Care Reform is a big issue now and many states are establishing their own programs and not waiting for government mandates. He stated that the New London County Medical Association held a meeting with their local legislators and stressed the fact that it is very important for the county associations to have these types of meetings.

He reported that the Building Committee was meeting, following the Council meeting, with three architectural firms to discuss preliminary planning and will select one of the firms to make a feasibility study.

Dr. Ahamed reported on his attendance at the AMA meeting and proposed that plans be promulgated to obtain greater exposure for our delegation, it was felt that only in this way can a Connecticut delegate obtain some meaningful post at the AMA level.

He reported that a meeting was held with Governor Weicker's health policy assistant and with Senator Lieberman and the CSMS Health Care Reform proposal was discussed.

Dr. Ahamed suggested that a meeting be held with the deans of the medical schools and the leadership of CSMS. It was VOTED to authorize the President to proceed to make such arrangements.

Report of the Executive Director

Mr. Norbeck reported on the following items:

(a) The AMA and the Minnesota Medical Association might challenge the health reform legislation which, among other things, prohibits physicians from showing the 2% tax on physicians' gross revenues as a line item on the bill to patients. In this way, the legislators manages to pass the blame for the tax increase on the physicians.

(b) Many physicians know that the California Medical Association had introduced Proposition 166 for the November election, a health reform proposal which would have required employers to pay at least 15% of basic health care for employees who work at least 17.5 hours per week. This proposal, which enjoyed a 20 point lead in the polls as late as August, eventually was defeated by the electorate in a 68-32 vote. An insurance company television blitz in early September and then again in the three weeks before the election buried Proposition 166.

(c) CBIA held "Managed Competition" conference on January 12 which featured Stanford Professor, Alain Enthoven, and two state regulators from Wisconsin and Pennsylvania. Mr. Norbeck mentioned that he was present at the conference and the lunch that followed and reported that there was a discussion and interest by the business community in hospital and physician-specific health care data and rating systems. He explained some of the problems involved in such systems.

(d) Ohio had recently passed mandatory assignment legislation with rather high income levels ($40,000). Mr. Norbeck also reported on a recent Gallup Poll of Congress which indicated strong support for a Medicare means test.

Report of the Auxiliary

Mrs. Joseph Czarsty, President of the Auxiliary, stated that her report was a sad one. That after 50 years they will be disbanding the Auxiliary. It was reported that there are only a few members left and they have tried everything possible to keep the Auxiliary going, but were not successful. A management firm was also hired to stimulate membership, but failed. She reported that Middlesex County, with 71 members, have been asked to be the link from Connecticut to the national office and has agreed and the national office will use that organization as a conduit into Connecticut and mail directly to a few members-at-large. The Middlesex County organization will be given all the privileges that are granted to a state organization and will be allowed one delegate and one alternate to national meetings. A final meeting will be held of the Auxiliary to determine the disposition of their funds. Mrs. Czarsty thanked the members of the Council for their moral and financial support and the staff for their cooperation. It was VOTED that a resolution should be prepared commending the Auxiliary for their efforts and that Mrs. Czarsty's name should be included in the resolution.

Legislative Update

Dr. Wetstone reviewed the following bills that were coming up in the Legislature:

Ownership of Licensed Pharmacies by Licensed Practitioner

Medicaid Reimbursement to Physicians

Establishment of a Health Care Task Force

An Act concerning Uncompensated Care Pool

Banning of Smoking in Public Buildings

Cost of Obtaining Medical Records from Health Care Providers

An Act Re Establishment of Medical IRA's

An Act to Repeal the Requirement to Provide Medical Services to Low Income Groups (i.e. Taking special health care programs not mandatory for physicians.)

An Act to establish a Quasi-Health Care Corporation to Control Health Care Costs

Establishing a Task Force on the Donation of Services for the Medically Uninsured

Removing of Income Limits for Medicare Assignment

An Act prohibiting Coercing a Person into Dying

An Act concerning the Composition of the Connecticut Medical Examining Board

Requirement of Motorcyclists to Wear Protective Headgear

An Act concerning Confidentiality of Personal Medical Records, Disease

Reporting, Immunization and Aids

Dr. Wetstone stated that these are just a few of the many bills that CSMS is tracking and watching very carefully.

Report of AMA Delegation

A summary report of the AMA meeting held in Tennessee in December was distributed to members of the Council with the agenda. Major issues covered in the report were: Health System Reform, Managed Care, Global Budgeting, Health Access America, Physician Ownership of Medical Facilities, Professional Liability Issues, etc. Dr. Beck reported he attended a meeting where the President-Elect of the AMA spoke and reassured the group that the AMA is "on the Job" in Washington, D.C. and that AMA had met with President Clinton during his campaign and also with the transmission team. Meetings will be also held with Hilliary Clinton, Chairperson of the President's Task Force on National Health Reform.

Dr. Kamens submitted a written report stating he was requested to attend a special meeting between Health Care Financing Administration representatives and some of the leadership of the AMA. The meeting was designed to inform the AMA about the progress being made with regard to the new Health Care Quality Improvement Initiative and to provide AMA with an opportunity to express concern about the algorithms of care utilized in the Uniform Clinical Data Set System (UCDSS) for conducting computerized review. As an AMA delegate/PRO medical director of a pilot PRO, Dr. Kamens was asked to demonstrate the role that practicing physicians play in the algorithm modification process. The Connecticut process was demonstrated and received favorable comment at the end of the meeting. It was VOTED to accept these reports as information.

Committee on Perinatal Morbidity and Mortality

(a) Acting on a recommendation of subject committee, it was VOTED that CSMS support the State of Connecticut's efforts toward full immunization and push for full government funding to allow the state to purchase the serum for all legally required pediatric immunizations.

(b) A representative from the Department of Health Services presented to the Committee a capacity survey that was done in conjunction with the Baby your Baby Campaign, which is a two year awareness program for prenatal care that was launched this summer. The Baby your Baby Campaign encourages pregnant women to call an 800 number for a referral to prenatal care. The survey results showed that for a complicated case, the turnaround time was 24 to 48 hours to connect the mother with the care. Access problems were found such as bad experiences at clinics discouraged return visits, transportation, language and lack of continuity at the site of care. Acting on the Committee's recommendation, the Council VOTED that CSMS assist the Baby your Baby Campaign by recruiting members physicians willing to take a limited or unlimited amount of referrals from the 800 line.

Connecticut Electric Data Interchange (EDI) Project

It was VOTED to endorse the Connecticut Electronic Data Interchange Project. The EDI project is a partnership of insurance payers, providers and others that is working toward the complete electronic transmission of medical claims in the state. The goal is to create one integrated and interactive electronic network for the state that would include eligibility determination, claim processing and electronic transfer of payment.

Recognition Award

The Committee to Study Perinatal Morbidity and Mortality informed the Council that they would like to establish an annual recognition award that would be presented at the Connecticut State Perinatal Education Conference held each year. The award would be presented to a physician in the state who had made a significant contribution in the field of obstetrics or pediatrics. The committee would be responsible for accepting nominations and granting the award. The recipient would receive an engraved plaque. During discussion, the opinion was expressed that there are many CSMS committees and it would be not wise to establish an award in one specialty and that further study was needed in the area of granting awards. It was VOTED to table a motion that CSMS establish a Physician's Recognition Award for an outstanding physician in the Society.

YOCON®
YOHIMBINE HCI

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85

AVAILABLE AT PHARMACIES NATIONWIDE

**PALISADES
PHARMACEUTICALS, INC.**
219 County Road
Tenafly, New Jersey 07670
(201) 569-8502
1-800-237-9083

Workers' Compensation Medical Fee Advisory Group

A communication from Marvin Arons, M.D., Chairman, accompanied by a report from Jeannette C. Schreiber, Esq. was distributed to the Council. Attorney Schreiber joined the Council to discuss the documents. The summary gave an overview of the activities of the group to date. It was stressed that the documents were to be kept confidential for the present time and were to be collected following discussion of the subject. Following discussion, she reported that she was very pleased at their progress and are building some good relationships. Also, it was reported that several additional meetings were necessary to conclude the issue.

Immunization Survey

A communication from David Black, M.D., M.P.H., a pediatrician at the University of Connecticut and the American Academy of Pediatrics representative on the State of Connecticut's Immunization Advisory Council, is currently undertaking a survey of physicians' office to determine areas of low immunization coverage to concentrate the state's resources in the coming year. The study is being endorsed by the Department of Health Services and the Connecticut Academy of Pediatrics and CSMS is requested to endorse this effort. It was VOTED to endorse the study.

Dates of Council and House of Delegates Meetings

It was VOTED to approve the following dates:

Council: Wednesday, February 24, 1993

Council: Wednesday, April 7, 1993

House of Delegates:

May 12, 1993, Ramada Inn, Meriden.

"Where Have All the Children Gone"? Inpatient Pediatric Care in Connecticut

JOHN D. THOMPSON, R.N., M.S., KEON-HYUNG LEE, M.P.H., JOHN T. LYNCH, M.P.H., AND DONNA DIERS, M.S.N.

ABSTRACT—Admission of children to hospitals in Connecticut dropped precipitously from 1981 to 1991. Regionalization of pediatric inpatient care is happening without plan. Connecticut data show a change in case-mix of pediatric cases, especially in surgical services and child mental illness categories. Planning for pediatric inpatient services should include considerations of case-mix, cost, and especially, quality of care in general hospital pediatric units with very low volume.

Introduction

IN 1988, while assessing the effect of the DRG prospective payment system on the number of admissions and inpatient days in Connecticut's general hospitals, a dramatic drop in these two indicators was noted in the pediatric services. The observation was made, "Although pediatric days and admissions are also dropping, this is part of a long-term trend, while adult medical-surgical days and admissions only started to drop in 1983. ..."[1]

A few statistics will support the observation. In 1973, 183,444 days of care were rendered on the pediatric services of 34 general hospitals in Connecticut. By 1991 only half as many (90, 319) days of care were given in the 32 general hospitals offering pediatric inpatient services.* More importantly, in 1979 six hospitals had an average daily pediatric census of fewer than five patients; in 1991, 20 out of the 32 hospitals had this low census. Nor is this a phenomenon limited to Connecticut hospitals; similar, though not quite so dramatic declines were noted by Perrin in 1986[2,3] and Kozak in 1987.[4] More recent comparisons of this drop in admissions and patient days are illustrated in Figs. 1 and 2. Children are not being admitted to or treated in Connecticut's hospitals at anywhere near the previous rate.

The implications of these changes should be explored now because a major change in the provision of inpatient pediatric care for the state is underway: the building of two children's hospitals, one in New Haven in conjunction with the Yale University School of Medicine, and the other in Hartford with the Hartford Hospital and the University of Connecticut School of Medicine. Outpatient care is of primary importance in pediatric services. But how can planning best be projected to optimize the contributions of all Connecticut's hospitals to the inpatient care of children?

The patterns of hospitalization were examined in a series of studies of the volume of services by differing pediatric populations, by diagnoses, and by type of hospital. Three data sets were used: the Patient Census Reports of the Connecticut Hospital Association; Pediatric Admissions by Patient Origin, published in 1990 by the Connecticut Health Information Management Exchange (CHIME); and a 100% sample of pediatric discharges (ages 14 and under) from all Connecticut hospitals in fiscal years 1981 and 1991. Data for the pediatric services of general hospitals only were examined for these years; data from the one special children's hospital

---

The late JOHN D. THOMPSON R.N., M.S., Professor Emeritus, Department of Epidemiology and Public Health and School of Nursing, Yale University; KEON-HYUNG LEE, M.P.H.; JOHN T. LYNCH, M.P.H., Vice President for Research, Connecticut Hospital Association; DONNA DIERS, R.N., M.S.N., The Annie W. Goodrich Professor, Chair, Adult Health Division, School of Nursing, and Lecturer, Department of Epidemiology and Public Health, Yale University.

The authors are grateful for the comments of Lawrence Scahill, R.N., M.S.N., M.P.H., and Joseph Warshaw, M.D., on an earlier draft of this article.

*St. Mary's Hospital in Waterbury, though operating major pediatric outpatient services in conjunction with the Waterbury Hospital, supported the plan that all inpatient admissions would be to the pediatric service of the latter institution. Memorial Hospital in Meriden began to phase out its pediatric inpatient service shortly after Meriden-Wallingford and World War II Memorial Hospitals merged.

Figure 1.
Admissions
All Connecticut General Hospitals
Cumulative Percent Change
Pediatric and Adult Medical-Surgical
1979 Base Year
FY 1980-1991

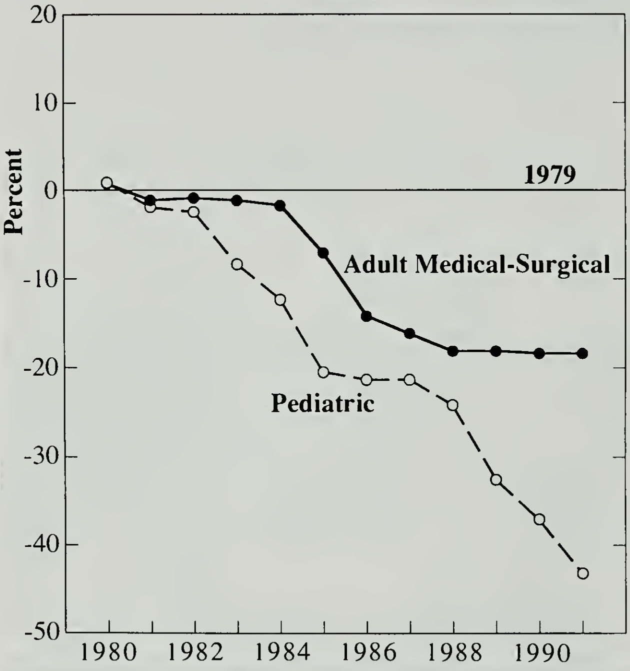
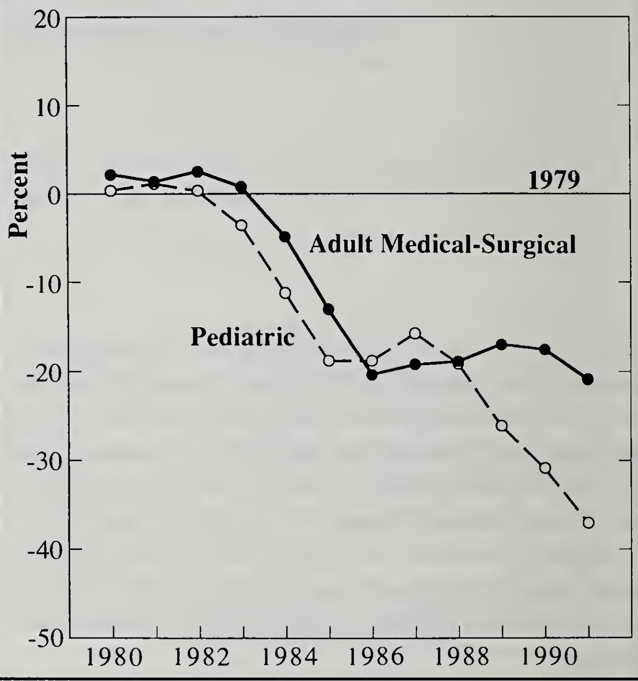


Figure 2.
Patient Days
All Connecticut General Hospitals
Cumulative Percent Change
Pediatric and Adult Medical-Surgical
1979 Base Year
FY 1980-1991



(Newington Children's Hospital) were not used. Newborn diagnoses were discarded from the 100% samples of 1981 and 1991 data since these children are usually cared for in nurseries or neonatal intensive care units in Connecticut, not in pediatric services.

Discharge Rates by Population

A total of 29,120 discharges were recorded for FY 1981 and 18,610 for FY 1991, a discharge rate of approximately 45/1,000 for 1981, and 30.2/1,000 for 1991, based on the 1980 and 1990 census. Using the CHIME data set pediatric admission rates were compared across the nine Connecticut Hospital Market Areas developed by Legnini.[5] Admissions per thousand in this data set varied among the areas from 13.52/1,000 to 20.96/1,000 population ages 19 and below.* Two patterns were discernable in the data. The relative risk of admission was higher in those towns where hospitals were located. Admission rates were significantly higher in core cities compared to the rest of the state.

While there was a significant difference in total pediatric admission rates between 1981 and 1991, there were no dramatic differences in the 1991 admission patterns across the state except in the older core city urban areas each of which was the setting of one or more general hospitals.

Case-Mix

An attempt was made to examine the differences in case-mix of the pediatric inpatient services (age 14 and under) for FYs 1981 and 1991. Table 1 lists the top 10 DRGs by number of pediatric discharges for the two years. These DRGs accounted for 49.4% of discharges in 1981 and 44.1% in 1991, and are used to illustrate the total case load of all hospitals.

The differences in case-mix are dramatic so far as surgical patients are concerned, but remarkably similar in the ranking of most medical pediatric admissions.

The surgical DRGs accounted for over one-third of the discharges in the top 10 DRGs in 1981, while the one surgical DRG in 1991 accounted only for 4.5% of that

---

*The disagreement across the data sets on admissions or discharges per thousand are due to three factors: 1) different age limits in the definition of pediatric patients across hospitals, 2) the inclusion of psychiatric patients, and 3) the population base used, ie, U.S. Census or Connecticut's precensus projections.

Table 1.

The Top 10 DRG Discharges for Patients Age 14 and Under:
All Connecticut Hospitals, Fiscal Year 1981

DRG	Title	Discharges		Rank	
		FY '81	FY '91	FY '81	FY '91
60	Tonsillectomy and/or adenoidectomy only, age 0-17	2,759	313	1	11
98	Bronchitis and asthma, age 0-17	2,489	2,203	2	
184	Esophagitis, gastroenteritis, and miscellaneous digest disorders, age 0-17	2,452	1,277	3	2
58	T and A procedures except tonsillectomy and/or adenoidectomy, age 0-17	1,313	15	4	137
91	Simple pneumonia and pleurisy, age 0-17	1,285	935	5	4
70	Otitis media and URI, age 0-17	942	404	6	7
163	Hernia procedures, age 0-17	878	212	7	18
422	Viral illness and fever of unknown origin, age 0-17	846	966	8	3
26	Seizure and headache, age 0-17	759	565	9	6
33	Concussion, age 0-17	662	158	10	31
Total Top 10 DRGs		**14,386**	**7,048**		
Total Discharges		**29,120**	**18,610**		

The Top 10 DRG Discharges for Patients Age 14 and Under:
All Connecticut Hospitals, Fiscal Year 1991

DRG	Title	Discharges		Rank	
		FY '91	FY '81	FY '91	FY '81
98	Bronchitis and asthma, age 0-17	2,203	2,489	1	2
184	Esophagitis, gastroenteritis, and miscellaneous digest disorders, age 0-17	1,277	2,452	2	3
422	Viral illness and fever of unknown origin, age 0-17	966	846	3	8
91	Simple pneumonia and pleurisy, age 0-17	935	1,285	4	5
298	Nutritional and miscellaneous metabolic disorders, age 0-17	814	323	5	17
26	Seizure and headache, age 0-17	565	759	6	9
70	Otitis media and URI, age 0-17	404	942	7	6
167	Appendectomy w/o complicated principle diagnosis, age <70 w/o complications/comorbidites	371	645	8	11
451	Poisoning and toxic effects of drugs, age 0-17	350	355	9	16
71	Laryngotracheitis	325	386	10	13
Total top 10 DRGs		**8,210**	**10,482**		
Total discharges		**18,610**	**29,120**		

Table 2.—Discharges AMA* by MDC† 19, hospital group, and year

	1981		1991	
	MDC 19	All Discharges	MDC 19	All Discharges
Hospital Group A		1 (0.0%)		4 (1.1%)
Hospital Group B	11 (7.7%)	17 (0.1%)	16 (3.3%)	11 (0.1%)
Hospital Group C	4 (2.7%)	28 (0.2%)	3 (1.0%)	20 (0.4%)

*against medical advice †mental disorders

year's discharges in the top 10 DRGs. Ear, nose, and throat procedures took a precipitous drop in volume over the 10 year period, further extending the findings of Perrin, et al in their study covering the years 1972 to 1981.[2] Because of lack of data on ambulatory surgery activity for 1981 and 1991, it is impossible to say how much of this decrease is due to the fact that fewer tonsillectomies are being performed altogether or whether many are being done in ambulatory surgery settings. Both changes are probably contributing to this effect on inpatient pediatric utilization.

Examination of the case load of the medical DRGs is complicated by interpreting different admission rates for different-sized populations. The first ranked DRG in 1991, Bronchitis and Asthma, for example, accounted for a higher percentage of the 1991 discharges (11.83%) than of the discharges in 1981 (8.5%). Discharges per thousand children were slightly higher in 1981 (3.84) than in 1991 (3.75), but not as dramatically different as in other most frequently admitted medical diagnoses. DRG 184 (Esophagitis), for example, had a discharge rate per thousand of 3.79 in 1981, and 2.17/1,000 in 1991; while simple pneumonia reflected a discharge rate of 1.99/1,000 in 1981 and 1.59/1,000 in 1991.

The surgical procedures in the top 10 were replaced in the 1991 ranked list by Nutritional and Metabolic Disorders, Toxic Effects of Drugs and Laryngotracheitis while the six medical disorders in the top 10 in 1981 remained in the top 10 in 1991 with little change in relative position. Notable is the finding that in 1991, within the top 10, the four DRGs dealing with conditions of the respiratory system account for 21% of all pediatric inpatient discharges.

Experience in Three Categories of Hospitals

In order to determine the variation in case load and mix between different types of hospitals, the discharge diagnoses for patients in three categories of hospitals were examined.

Hospitals in Group A were the two primary university hospitals. Those hospitals with an average daily pediatric census of less than five (21 in 1981, 20 in 1991) were considered as Group C. The remaining hospitals, most of which have some type of affiliation with one or other of the medical schools comprised Group B. These three groups of hospitals had quite different volume changes between 1981 and 1991. The university hospitals accounted for 24.3% of pediatric patient days in 1991; patient admissions increased 10.9% over the 10 years, and patient days increased by the same percentage. The pediatric patient days in the smaller hospitals (Group C) dropped 61.6% and decreased 29.3% in the Group B hospitals, with a similar drop in admissions in both types of hospitals.

There were, however, some trends across all hospitals over the 10-year period. All hospitals demonstrated a change in average pediatric case mix index. The university hospitals' index for the first 30 DRGs was 0.9481 for 1991, an increase of 5% over the 10-year period. The case mix for the first 30 DRGs for Group C hospitals rose to 0.5208 from 0.4317 (17%), and Group B hospitals' average case weight rose to 0.5373 from 0.4878 (9%).

Mortality as an index of hospital quality has been called into question.[4] Since we are concerned with children, where mortality ought to be low to nonexistent, it is of

Table 3.

The Top 10 DRG Discharges for Patients Age 14 and Under:
University Hospitals (Group A), Fiscal Year 1991

DRG	Title	Discharges		Rank	
		FY '91	FY '81	FY '91	FY '81
98	Bronchitis and asthma, age 0-17	396	195	1	2
422	Viral illness and fever of unknown origin, age 0-17	201	111	2	7
298	Nutritional and miscellaneous metabolic disorders, age 0-17	159	100	3	9
410	Chemotherapy	159	127	3	6
184	Esophagitis, gastroenteritis, and miscellaneous digestive disorders, age 0-17	134	209	5	
109	Cardiothoracic procedures w/o pump	127	25	6	40
26	Seizure and headache, age 0-17	126	137	7	3
405	Lymphoma or leukemia, age 0-17	120	61	8	16
91	Simple pneumonia and pleurisy	116	99	9	10
467	Other factors influencing health status	93	45	10	26
Total Top 10 DRGs		**1,722**	**1,109**		
Total Discharges		**4,512**	**4,069**		

The Top Ten DRG Discharges for Patients Age 14 and Under:
Smaller Connecticut Hospitals (Group C), Fiscal Year 1991

DRG	Title	Discharges		Rank	
		FY '91	FY '81	FY '91	FY '81
98	Bronchitis and asthma, age 0-17	542	1,211	1	2
184	Esophagitis, gastroenteritis, and miscellaneous digestive disorders, age 0-17	327	1,070	2	3
91	Simple pneumonia and pleurisy	301	650	3	5
298	Nutritional and miscellaneous metabolic disorders, age 0-17	247	80	4	26
431	Childhood mental disorders	191	32	5	55
422	Viral illness and fever of unknown origin, age 0-17	174	357	6	7
167	Appendectomy w/o complicated principle diagnosis, age <70 w/o complications/comorbilities	166	318	7	9
60	Tonsillectomy and/or adenoidectomy only, age 0-17	133	1,306	8	
426	Depressive neuroses	121	15	9	87
26	Seizure and headache, age 0-17	110	260	10	11
Total Top 10 DRGs		**2,312**	**5,199**		
Total Discharges		**4,454**	**11,591**		

interest to examine hospital groups for mortality rates. In all three groups of hospitals, overall mortality in pediatric cases dropped from 1981 to 1991: Group A: 2.1% to 1.1%; Group B: 0.4% to 0.3%; Group C: 0.1% to 0.01%.

The data available include another potential measure of hospital performance—discharges against medical advice (AMA), which could be taken as an index of parent dissatisfaction, though we are not aware that this datum has been used for this kind of analysis before.

Table 2 shows the relationship between hospital group discharges AMA for 1981 and 1991. For this analysis, cases in MDC 19 (Mental Disorders) are shown separately.

The difference between cases in MDC 19 and others will be discussed later.

If the volume and case-mix of 1991 for the university hospitals (Group A) had represented patients hospitalized with the 1981 length of stay, the average length of stay for the top 30 DRGs of 1991 would have been 6.14 days rather than the actual of 4.5 days. This represents a 26.7% decrease in length of stay. For the other two groups, this same decrease would have been 58% for the Group C hospitals and 22.7% for the Group B hospitals.

Case-Mix by Hospital Type

What is of interest here is a more specific examination of the case-mix of the three hospital groups in 1991 to see if these hospitals are treating the same or different kinds of patients.

Two important observations can be made by examining the top 10 DRGs in the three hospital categories. Table 3 compares the university hospitals with the Group C hospitals. The first observation is that all three hospital categories are treating the same case load of the very high volume DRGs; the second is the emergence of hospitalizations for childhood mental illness as a factor in case mix.

For all three hospital categories, the most frequent reason for admission is Bronchitis and Asthma; the next medical conditions on the 1991 combined list (Table 1) are in the top 10 of each hospital category. The university hospitals are also treating the high incidence cases in their communities as well as providing specialized care for complex, rarely encountered cases. The different roles of the three types of hospitals are elicited from the remaining five ranked DRGs. For the university hospitals they are cardiothoracic procedures and childhood neoplasms, probably referrals. DRG 467 (ranked 26th in 1981, 10th in 1991) is shorthand for negative findings on a variety of workups for congenital and childhood conditions suspected through histories or screening findings, also referrals. DRGs for the Group B and Group C hospitals, the remaining five DRGs, except for the two discussed below, are for the hospitalization of less complicated childhood conditions.

DRG 422, Viral Illness and FUO, moves from rank seven in 1981 to two in 1991 in university hospitals, and moves from rank seven to six in Group C hospitals over the 10-year period. The DRG is essentially "rule out sepsis." University hospitals are getting these cases as referrals. Without further analysis by DRG and length of stay, it is not possible to determine whether the small hospitals that appear to have a higher volume of these cases in 1991 are actually transferring them, but it would be logical to assume so.

One would anticipate that the shift in case-mix away from the schedulable surgery DRGs to the acute medical conditions would result in a change in the source of pediatric admissions from scheduled to emergency. In fact, that happened, but other changes happened as well.

In 1981, 99% of discharges were to home in Group A hospitals; in 1991, 90.7% were and the difference is accounted for primarily by discharges to "other" institutions, not general hospitals. Newington would be the primary receiver. In 1981, 82.4% of discharges were to home in Group B hospitals; in 1991, 70.4% were, but the difference here is not only transfers but discharges to home health care. In Group C hospitals in 1981, 71.3% were discharges to home; in 1991, 77.5% were, and this difference is nearly entirely accounted for by transfers to other hospitals or health care facilities.

In 1981 Medicaid was the primary payor source for 30.2% of pediatric discharges from Group A hospitals; in 1991 that percent was 34.5. In 1981 Medicaid paid for 16.5% of hospitalizations in Group B hospitals; in 1991 that figure was 35.8%, more than double. In 1981 Medicaid was the primary payor for 20.6% of discharges from Group C hospitals; in 1991 that figure was 25.3%.

On the other hand, it is interesting to note that in 1991 all three groups of hospitals had about the same level of HMO-covered clientele (HMO was not an option in 1981); Group A: 11%; Group B: 11.8%; Group C: 12.2%.

Mental Diseases and Disorders

The major finding in the smaller hospitals' case-mix is the two categories of mental disease and disorders. The notable numbers of discharges in DRGs 431, Childhood Mental Disorders and 426, Depressive Neuroses, in the smaller hospitals is in some way due to the design of the patient sample and in the pattern of the provision of children's mental health services in Connecticut.

There were at the time of the study four dedicated inpatient and special psychiatric services for children in the state's general hospitals for which data were available. One unit each was present at St. Raphael's and Yale-New Haven in New Haven, one at Mount Sinai in Hartford, and one at Newington Children's Hospital. Data for these discharges in three hospitals (Newington excluded) were not classified as part of the pediatric census but treated in the psychiatric service. The most active of these services was at Mount Sinai which had a small pediatric service in 1991 and was categorized as one of the Group C hospitals but treated many children with mental illness. This accounts for the appearance of these DRGs in the small hospital case-mix list (Table 2).

When the patient sample was drawn, age (14 and below) rather than clinical service was the indicator for inclusion. Therefore, younger psychiatric admissions were included for the 1981 and 1991 regardless of what service they were treated on within the hospital.

To understand this major change in hospitalization patterns, data on patients hospitalized in Newington Children's Hospital were reintroduced into the data set. This brought the number of MDC 19 (Mental Disorders) patients admitted to Connecticut's hospitals in 1991 up to 1,023 admissions as compared to 502 in 1981. In 1991, 31.9% of discharges with mental illness were from the four special units noted above.

Unfortunately, psychiatric DRGs are weak predictors of treatment complexity or resource use, but when some secondary patient data are examined, there are indications that there are other differences between the 1981 and 1991 as important as the volume increase.

The number of pediatric psychiatric admissions from emergency rooms increased from 18.9% in 1981 to 34.9% in 1991; further, the number of transfers from other hospitals of MDC 19 patients increased from 4.1% to 7.1%. More important, the number of MDC 19 patients covered by Medicaid jumped from 7.3% in 1981 in Group A hospitals to 51.7%; in Group B hospitals the change was from 34.5% to 46.2%; in Group C, from 35% to 43.1%.

Discussion

What do all these changes predict for the future of pediatric hospital services in Connecticut? The first and most obvious finding (though not the best documented) is that hospital centered pediatric practice has already switched to the outpatient department, particularly to ambulatory surgery, private referred ancillary services, and the emergency service.

Regionalization of children's services is already happening in Connecticut, without plan, oversight, or rationale. The implications for both the referring and referred-to hospitals are serious.

Treating fewer, sicker, poorer children primarily in the medical pediatric services, requiring a complex pattern of services, and a full array of follow-up options available on discharge, translates into a new type of pediatric hospital care, especially in the receiving hospitals.

In addition to patient characteristics, there are three other factors which should influence the future design of inpatient care: referral patterns, the hidden cost of pediatric inpatient services, and the issue of quality of patient care.

These data show clearly that the university hospitals are receiving increasing numbers of referrals, especially from smaller institutions. With the formalization of two children's hospitals, each the primary teaching unit of a university, one can predict further issues for other hospitals since the university hospitals treat both the routine high volume cases as well as the rarer conditions requiring specialty care.

Inpatient pediatric care is no longer T and A's, hernia repairs, infectious diseases, and the occasional dumping of mildly ill children on the nursing staff while their parents party. Physicians hospitalize children as infrequently as possible. Antibiotics and payment systems have created an environment in which children who now are hospitalized truly *need* that resource.

The design of the present study excluded children over the age of 14. One suspects that the patterns detected here would only be reinforced were children between 14 and 17 (by DRG definitions) or even older were included. Chronic illness in children (cystic fibrosis, for example) no longer kills before maturity. Children with spina bifida, with Down's syndrome, with the various now-treatable congenital anomalies survive into their late teens and beyond. And Connecticut will soon face the problem of caring for children born of HIV-positive parents, the majority of whom in this state are substance abusers. These children, even when they are not HIV-positive have enormous social and medical problems that require specialized services not always available in small hospitals.

Although the matter of the hidden costs of pediatric inpatient care was not dealt with in the present inquiry, two previous studies of the costs of nursing care in the clinical services of these same Connecticut hospitals reported higher costs for routine nursing care on pediatric inpatient services.[7] The costs for nursing in pediatric units (not neonatal intensive care units, pediatric intensive care units, or newborn special care units) was 52.9% of room and board; in general adult medical-surgical units, the nursing cost was 49.1% of room and board. The referenced study did not separate hospitals by the groups used here. In another study when nursing weights for DRGs were estimated by a peer group of nursing experts, increased nursing time was projected for selected DRGs when pediatric patients were being treated.[7]

The reason that these increased expenses are called hidden costs is because most hospitals do not isolate their nursing costs by clinical service. The cost differential between nursing services in pediatrics and adult medical surgical are there; however, the amount of cost shifting will never be known until finer microcosting is carried out. The problem of using an economies-of-scale analysis for the provision of pediatric inpatient services is the fact, as demonstrated by this study, that large hospitals treat a different case-mix from small hospitals.

To the extent that nursing costs are a factor here, that issue is complicated in a rate-setting environment that does not analyze data separately for children in DRGs undefined by age, and in which nursing data are not available except in special studies. If pediatric care is indeed being regionalized, some consideration should be given to recognizing in the payment structure those hospitals that are increasingly becoming the receivers of very ill children, where the costs will increase not only because of the diagnostic and treatment problems, but because children are, by definition, more dependent and thus require more nursing time, hours, and dollars.

As difficult as isolating cost implications, the identification of quality differences between the three classes of hospitals is even more troublesome.

The Accreditation Manual for hospitals of the Joint Commission on Accreditation of Health Care Organizations (JCAHO) contains but a few explicit standards for pediatric inpatient services, relying on the general provision of age-appropriate care. The manual does state there should be separation of adults and children in intensive care units "when possible." There is a specific standard about the necessity for coordination of hospital services, specifically mentioning children's care involving the coordination of that care with parents and guardians, a variety of special social services, and a physical environment to provide comfort and security to the child. One wonders whether these standards are met for the 68% of the children with psychiatric diagnoses treated on adult units, or whether such services can be available for any children treated in general units with an average daily census of 2.1. The number of children discharged AMA is a particularly troubling statistic.

Recommendations

The data make clear the necessity of rationalizing regionalization of inpatient care for children in Connecticut. Four hospitals with an average daily census of less than five (the average of the four hospitals was 1.5 patients per day) are located in the same towns as hospitals with larger inpatient units. There is no reason for these small services to be maintained.

There are an additional six hospitals with small pediatric services with a travel time of less than 30 minutes from a large pediatric inpatient service. None of these 10 hospitals have neonatal intensive care units.

It is always difficult for a hospital to drop a clinical service, particularly a service with the image-enhancing potential of caring for children. Such an image contributes not only to the picture the community holds about its hospital, but effects the bonds that tie the hospital staff to their institution. It is held that these important identifiers can be retained by providing pediatric outpatient and ambulatory care units, which are the site of most pediatric care these days anyway.

Regionalization consists of two components: the merger of inpatient services where indicated, and the supportive planning for the maintenance of those small pediatric inpatient services that cannot be closed because of their geographic isolation. Both of these components must be accompanied by changes in the emergency transportation system and by a general education program for the communities effected by regionalization.

All these changes would be more acceptable if the decisions were made based on formal clinical criteria as well as on administrative guidelines. It is recommended that the Connecticut Academy of Pediatrics, the Connecticut Hospital Association, and the Connecticut Nurses Association, through a joint committee, set up the criteria for regionalization of inpatient pediatric services for the state, involving all of their constituencies in the process.

The experience of St. Mary's Hospital and Waterbury Hospital will be a valuable source of information as well as the recent phase-out in Meriden. There is some evidence that such closure decisions might occur unilaterally or be recommended by a public body; these kinds of decision processes would imperil the successful design of a regional system for the care of children in Connecticut.

The other major problem, the care of children with mental illness, is less well delineated but just as important. What is needed here are clearer clinical recommendations about the desirable patterns of care, including the role of the inpatient service in the treatment of these conditions. The Connecticut Academy of Pediatrics, the Connecticut Association for Child Psychiatry, and the Connecticut Association of Advocates for Child Psychiatric Nursing should, together with the State Department of Mental Health, generate a set of recommendations involving a variety of treatment settings including specialty hospitals, general hospitals, and state institutions for the care of children with mental illness.

REFERENCES

1. Thompson JD: DRG prepayment — its purpose and performance. *Bulletin of the New York Academy of Medicine* 1988; 64:25-51.

2. Perrin JM, Valvon J, Sloan FA: Changing patterns of hospitalization for children requiring surgery. *Pediatrics* 1986; 77:587-92.
3. Perrin JM, Homer CJ, Berwik DM, Woolf AD, Freeman JL, Wennberg JE: Variations in rates of hospitalization of children in three urban communities. *N Engl J Med* 1986; 320:1183-6.
4. Kozak LJ, Norton J: Hospital use patterns for children in the United States - 1983-1984. *Pediatrics* 1987; 80:481-90.
5. Legnini MW: Small-area variations in Connecticut hospital discharges. Ph.D. Dissertation, Yale University School of Medicine, Department of Epidemiology and Public Health, 1988.
6. Krakauer H, Bailey RC, Skellan KJ, Steward JD, Hartz AJ, Kuhn EM, Rimm AA: Evaluation of the HCFA model for the analysis of mortality following hospitalization. *Health Services Research* 27:317-36.
7. Thompson JD, Diers D: Nursing resources. In Fetter RB, ed, *DRGs: Their design and development*. Ann Arbor: Health Administration Press; 1991:144-5.

John D. Thompson devoted the bulk of his career to hospitals—their history, their management, their data, their structure, and their idiosyncrasies. He is best known for the work that became diagnosis Related Groups (DRGs), but his scholarship and teaching were much wider. He wrote on Florence Nightingale, on the history of st. Thomas' Hospital in London, on dialysis services and patients in Connecticut, on everything about hospitals from staff organization and relationships to how to get the food served hot and on time. He served in the Navy in World War II as a pharmacists' mate (men nurses were not granted officer status), an assistant administrator at Monefiore Hospital in New York, and as associate dean for Planning at the Yale School of Medicine (1968-74). But most of his career was in the Department of Epidemiology and public Health at Yale where he directed the Hospital Administration and later the Health Services Administration Division, a position he held at the time of his death at age 75 in August 1992. He was immediate past president of the Beaumont Medical Club at Yale at the time of his death.

"I'm practicing medicine the way I think it should be practiced, sans the paperwork and administrative overload."

Owen Brodie, MD, joined CompHealth's locum tenens medical staff in 1989, after 21 years in private practice. Since then he's worked in temporary assignments in state facilities, filled in for attending physicians, covered for private practitioners across the country.

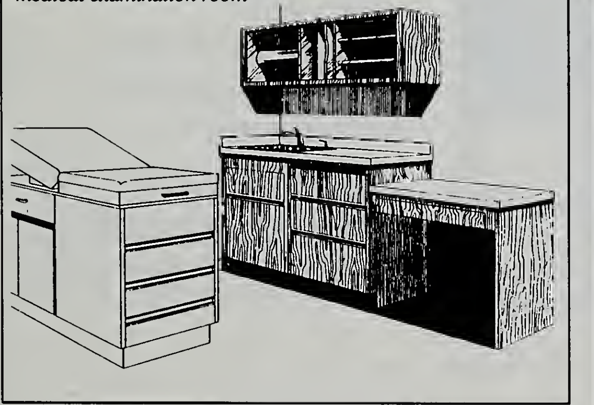
A pilot. A historian. A board-certified psychiatrist. Southern to a fault. Owen Brodie knows...

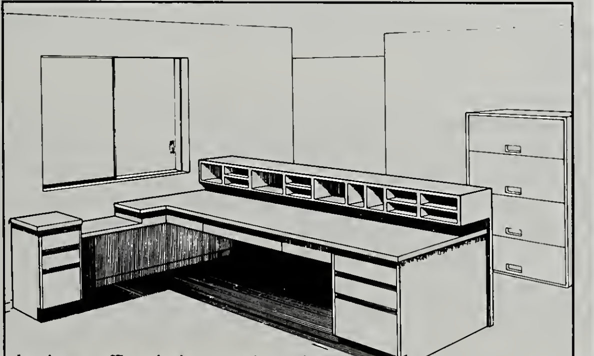
It's a great way to practice medicine

CompHealth
LOCUM TENENS

1-800-453-3030
Salt Lake City ■ Atlanta ■ Grand Rapids, Mich.

medical examination room





business office desk / nurse's station

MODULAR CABINETRY ★ DESK & FILE SYSTEMS

A Delta designed office can make you more successful. So whether you're planning to move or will be opening a professional office or are about to refurbish your current one, Delta can show you a variety of ways to succeed. Custom designed cabinetry and desk systems are matched to your standards of practice.

- Delta offers you the most comprehensive selection of cabinets and desk systems available anywhere!
- Delta offers planning and design analysis of your architect's or Dealers space plan at *no cost*.
- Delta's unlimited choices of finish and texture can match any decor theme.
- Delta designed modular cabinetry offers *special tax advantages*. Find out how you can benefit from our unique approach.
- Every office environment Delta creates or enhances is *guaranteed* to function more efficiently, more ergonomically and to look more "patient appealing".

NEW OFFICES
EXPANSION ★ REFURBISHMENT

CALL FOR FREE CONSULTATION

CALL FOR FREE ON-SITE SURVEY

delta enterprises, inc.
21 Appomattox Drive ■ Manalapan, NJ 07726-1801
(908) 536-3000 ■ Out of Town (800) 735-3358

Because One Size Doesn't Fit All...

Eli Lilly and Company can suit all your needs with the most complete line of human insulins available.

Featuring Humulin 70/30* and our latest addition to the premixed line, Humulin 50/50† —especially useful in situations in which a greater insulin response is desirable for greater glycemic control.

Humulin®
human insulin
(recombinant DNA origin)

Tailor-made options in insulin therapy

WARNING: Any change of insulin should be made cautiously and only under medical supervision.

* Humulin® 70/30 (70% human insulin isophane suspension, 30% human insulin injection [recombinant DNA origin]).

† Humulin® 50/50 (50% human insulin isophane suspension, 50% human insulin injection [recombinant DNA origin]).

Lilly

Global Excellence in Diabetes Care
Eli Lilly and Company
Indianapolis, Indiana
46285

HI-7918-B-349310 © 1993, ELI LILLY AND COMPANY

Tuberculin Reactivity Is Low Among Residents of a Connecticut Nursing Home

MICHAEL C. LINDBERG, M.D., DAVID WINSEMIUS, M.D., M.P.H., BERNADINE LAWYER, R.N., AND ROY V. ERICKSON, M.D.

ABSTRACT—Of 131 residents of a suburban New England intermediate and skilled nursing facility, 126 agreed to be evaluated by two-stage administration of purified protein derivative (5TU) for tuberculin reactivity. Only eight patients (6.4%) had a positive reaction, considerably fewer than reported by most previous studies. There were no statistically significant differences between reactors and nonreactors based in age, gender, or length of stay in the facility. There was a tendency toward an association of presence of risk factors and a positive response to tuberculin skin testing, but this did not achieve statistical significance (*P*=0.063).

SINCE 1985 the number of new cases of active tuberculosis have risen steadily.[1] Adults over the age of 65 account for proportionally more cases of active tuberculous infection than any group other than HIV infected patients.[2] The Centers for Disease Control (CDC) report an annual incidence rate for active tuberculosis of 21.5 per 100,000 community dwelling elderly. The incidence rises to 39.2 cases per year per 100,000 nursing home residing elderly persons.[3] However, the prevalence of positive tuberculin reactive status for residents of long-term care facilities has been reported as 9,200 per 100,000 residents to as high as 49,200 per 100,000 residents.[5]

It is estimated that 90% of cases of active tuberculous infection in elderly persons are due to reactivation of long dormant disease.[6] However, there are individuals who have cleared their previous infection, become immunologically naive, and are at risk for reinfection should they be exposed to a case of active tuberculosis.[7] For this reason it is crucial to identify the tuberculin-reactive status of all nursing home residents. Those who are tuberculin-positive can be monitored for reactivation of dormant disease. Those who are negative can be followed for skin-test conversion, especially after exposure to an active case of tuberculosis within the facility.[8]

This study was undertaken to assess the prevalence of positive reaction to tuberculin skin testing in a chronic care facility located in suburban Connecticut, and to see if the results were comparable to those defined in previous studies.

Methods and Materials

The study took place at a long-term care facility; there were 131 residents of the intermediate and skilled units, none having on record a previous positive tuberculin skin test or history of tuberculosis. Five tuberculin units per 0.1mL of purified protein derivative (Tubersol, Connaught) were introduced intradermally to the anterior forearm by the Mantoux technique. The test was read at 48 hours using the Sokol method.[9] A second identical test was administered two weeks after the first in those who had a negative (<10mm induration) response to the initial dose. A positive test was identified as >10mm of induration. The results were recorded.

Data concerning each resident were collected by chart review. Information was obtained regarding age, gender, race, length of stay in the facility, and presence of risk factors (Table 1) for the development of tuberculosis as defined by the CDC.[10]

MICHAEL C. LINDBERG, M.D., Fellow, Travelers Center on Aging, University of Connecticut Health Center, Farmington; DAVID WINSEMIUS, M.D., M.P.H., Assistant Medical Director, The McLean Home, Simsbury; BERNADINE LAWYER, R.N., Infection Control Practitioner, The McLean Home, Simsbury; ROY V. ERICKSON, M.D., Medical Director, The McLean Home, Simsbury.

Please address all correspondence to: Roy V. Erickson, M.D., Medical Director, The McLean Home, 75 Great Pond Road, Simsbury, CT 06070. (203) 658-2254.

Table 1.—Risk Factors for Tuberculosis
Silicosis
Gastrectomy
Jejunoileal bypass
≥10% below ideal body weight
Chronic renal failure
Diabetes mellitus
Corticosteroid therapy
Immunosuppressive therapy
Hematologic malignancy
Other malignancies
Abnormal chest radiograph

Statistical analysis was performed on the SYSTAT program using descriptive statistics, Student's t-test, and analysis of variance (ANOVA). A $P<0.05$ was considered significant.

Results

A total of 131 charts were reviewed. The characteristics of the patient population are summarized in Tables 2,3, and 4. The majority of residents were female, white, and over the age of 85. About one-half had been residents of the facility for greater than one year. Most had none of the risk factors listed in Table 1. Of the 131 residents, 126 agreed to have their tuberculin reactivity checked. Based on the two-step examination, eight residents (6.4%) were positive (Table 5). All positives were detected with the first test. Chest radiographs showed no evidence of active tuberculosis in the tuberculin responders.

An analysis of variance was performed to see if age had an effect on the prevalence of reactive tuberculin skin tests. No significant differences were identified ($P=0.903$) among the different age groups.

Student's t-test was used to determine if gender, length of stay, or presence of a risk factor for developing active tuberculosis influenced tuberculin reactivity. There were not enough data in this population to analyze for race. No significant differences were observed for gender ($P=0.861$) or length of stay ($P=0.463$). While the influence of risk factors on positive skin testing did not achieve statistical significance there was a tendency toward an association between the presence of a risk factor and a positive skin test ($P=0.063$).

Discussion

The results of this study show a considerably lower prevalence of positive reaction to tuberculin skin testing among residents of this facility than described in other studies. Table 6 summarizes the results of nine previous studies of tuberculin skin testing.[4,5,7,11-16] The prevalence of positive testing has varied from 9,216 to 49,233 per 100,000 residents depending on the facility studied.[4,5] The present study showed only a 6.4% positive response to testing (prevalence of 6,349 per 100,00). Even if the patients who declined to be tested were all reactive to tuberculin skin testing, the positive results would rise only to 9.9%, considerably lower than most of the reported studies.

Table 2.—Gender Distribution of Residents

	Female	Male	Total
Minimum Age	14	41	14
Maximum Age	105	98	105
Range (years)	91	57	91
Mean Age	85.1	83.1	84.7
Stand Dev.	11	12.3	11.8

Several explanations exist for the lower than expected positive rate. The small number of subjects in the current study gives less confidence in the results as population estimates. The majority of subjects were white women, the group previously defined as being at the lowest risk for tuberculous infection.[1] African Americans have approximately twice the risk of being infected with *Mycobacterium tuberculosis*,[17,18] men at higher risk than women.[1] African Americans were markedly underrepresented in the present study. Other groups at high risk for tuberculous infection not present in this patient population include Hispanics,[18] Native Americans,[19] immigrants from Asia and the Pacific Islands,[20] and those who have been homeless.[21]

In a region with a small high-risk population, the introduction of an index case of tuberculosis into the facility is less likely. Most of the nursing home residents

Table 3.—Age Distribution of Residents

Age	No.	Percent
<55	4	3.1
56-65	3	2.3
66-75	9	6.9
76-85	37	28.2
>85	78	59.5

Table 4.		
Race, Length of Stay, Presence of Risk Factors		
	No.	**Percent**
Race		
White	128	97
Black	3	2.3
Length of Stay		
<1 year	64	48.9
>1 year	67	51.1
Risk Factors		
Yes	51	38.9
No	80	61.1

have lived for a long time in New England and are relatively affluent. Of those foreign born, most are from western Europe; none are from Asia.

The low prevalence of tuberculin positivity in this study suggests a low risk for this patient population. Annual retesting of all current residents who are skin-test negative, as well as two-step testing of all new admissions will need to be done at all long term-care facilities.[8] Positive tuberculin reactors need be considered for prophylactic treatment with isoniazid if they are felt to be at high risk for the development of active tuberculosis.[10]

The results from our facility also need to be compared with those from other long-term care centers in the area. It will be necessary to follow tuberculin reactivity in facilities where there are more residents from minority populations, more admissions from urban areas, and more patients who have immigrated to the United States from nations with higher rates of tuberculous infection to allow a determination of the facilities at highest risk for cases of tuberculous infection.

Acknowledgement

The authors are grateful to Richard W. Besdine, M.D., for his review of and advice on the preparation of this manuscript.

Table 5.—Tuberculin Reactivity Status of Residents

PPD Reactive	n=126 **No.**	**Percent**
Status		
Negative	118	93.6
Positive	8	6.4

REFERENCES

1. Reider HL, Cauthen GM, Kelly GD, et al: Tuberculosis in the United States. *JAMA* 1989; 262:385-9.
2. Centers for Disease Control: Tuberculosis statistics in the United States, 1987. Atlanta; 1989, HHS publication no. (CDC) 89;8322.
3. Centers for Disease Control: Prevention and control of tuberculosis in facilities providing long term care to the elderly. Recommendations to the Advisory Committee for Elimination of Tuberculosis. *MMWR* 1990; 39:7-13.
4. Aronow WS: Tuberculosis screening in 1,161 elderly patients. *Chest* 1989; 96:961-2.
5. Alvarez S, Kasprzyk DR, Freundl M: Two-stage skin testing for tuberculosis in a domiciliary population. *Am Rev Respir Dis* 1987; 136:1193-6.
6. Stead WW, Dutt AK: Tuberculosis in elderly persons. *Ann Rev Med* 1991; 42:267-76.
7. Stead WW, Lofgren JP, Warren E, Thomas C: Tuberculosis as an endemic and nosocomial infection among elderly in nursing homes. *N Engl J Med* 1985; 213:1483-7.
8. Finucane TE: The American Geriatric Society statement on two-step PPD testing of nursing home patients on admission. *J Am Geriatr Soc* 1988; 36:77-8.
9. Sokol JE: Measurement of delayed skin test responses. *N Engl J Med* 1975; 293:501-2.
10. Centers for Disease Control: Screening for tuberculosis and tuberculous infection in high-risk populations and the use of preventive therapy for tuberculous infection in the United States. Recommendations of the Advisory Committee for the Elimination of Tuberculosis. *MMWR* 1990; 39:1-7.
11. Simon JA, McVicker SJ, Ferrell CR, Payne CB: Two-step tuberculin testing in a veterans domiciliary population. *S Med J* 1983; 76:866-70.
12. Welty C, Burstin S, Muspratt S, Tager IB: Epidemiology of tuberculous infecton in a chronic care population. *Am Rev Respir Dis* 1985; 132:133-6.
13. Dorkin E, Grzybowski S, Allen EA: Significance of the tuberculin skin test in the elderly. *Chest* 1987; 92:237-40.
14. Barry MA, Regan AM, Kunches LM, et al: Two-stage tuberculin testing with control antigens in patients residing in two chronic disease hospitals. *J Am Geriatr Soc* 1987; 35:147-53.
15. Gordin FM, Perez-Stable J, Flaherty D, et al: Evaluation of a third sequential tuberculin skin test in a chronic care population. *Am Rev Respir Dis* 1988; 137:153-7.
16. Creditor MC, Smith EC, Gallai JB, et al: Tuberculosis, tuberculin reactivity, and delayed cutaneous hypersensitivity in nursing home residents. *J Gerontol* 1988;43:M97-100.
17. Centers for Disease Control: Tuberculosis in Blacks— United States 1985. *MMWR* 1987; 36:212-20.
18. Centers for Disease Control: Tuberculosis among Hispanics-United States 1985. *MMWR* 1987; 36:568-9.
19. Centers for Disease Control: Tuberculosis among American Indians and Alaska natives-United States 1985. *MMWR* 1987; 36:493-5.
20. Centers for Disease Control: Tuberculosis among Asians/Pacific Islanders-United States 1985. *MMWR* 1987; 36:331-4.
21. Centers for Disease Control.:Tuberculosis control among homeless populations. *MMWR* 1987; 36:257-60.

(SEE TABLE 6 NEXT PAGE)

Table 6.—Nine Previous Studies of Tuberculin Testing, 1983-89

Study*	Facility Type	Geographic Location	Number Screened	Positive after first step† No. (%)	Positive after second step† No. (%)
Simon[11] 1983	Veterans Hospital Domicil. Unit	Dayton, OH	740	291 (39.3)	346 (46.7)
Welty[12] 1985	Veterans Hospital	Brockton, MA	588	139 (23.6)	184 (31.3)
Stead[7] 1985	Long-term Care	Arkansas	25,637	3,185 (12.4)	3,804 (14.8)
Alvarez[5] 1987	Veterans Hospital Domiciliary Unit	Johnson City, TN	587	230 (39.2)	289 (49.2)
Dorkin[13] 1987	Long-term Care	Vancouver, Canada	933	232 (29.4)	242 (25.9)
Barry[14] 1987	Chronic Disease Hospitals	Long Island, NY	347	109 (31.4)	121 (34.9)
Gordin[15] 1988	Veterans & Urban Hospital	Washington DC; West Virginia; San Francisco, CA	1,726	477 (27.6)	635 (36.8)
Creditor[16] 1988	Long-term Care	Chicago, IL	473	121 (25.7)	140 (29.6)
Aronow[4] 1989	Long-term Care	New York, NY	1,161		107 (9.2)

*For complete citations, see the reference list.

†Includes those with a prior positive PPD or positive history of tuberculosis .

Important Advances in Clinical Medicine

Anesthesiology

The Council on Scientific Affairs of the California Medical Association presents the following inventory of items of progress in anesthsiology. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome, and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, researchers, and scholars to stay abreast of these items of progress in anesthesiology that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Anesthesiology of the California Medical Association, and the summaries were prepared under its direction.

Transesophageal Echocardiography

TRANSEOPHAGEAL echocardiography offers a new perspective on the heart. With the introduction of smaller, more sophisticated transducers and the addition of color-flow Doppler and biplanar imaging, transesophageal echocardiography has become widely accepted as a low-risk method for the rapid, accurate examination of myocardial function, valvular disease, and aortic anatomy.

In skilled hands, transesophageal echocardiography is considered safe, with a low (< 1%) incidence of complications such as arrhythmias, hypotension, bronchospasm, and aspiration. The most catastrophic complication, esophageal rupture, is extremely rare and has been reported only in patients with esophageal disease or carcinoma; transesophageal echocardiography is contraindicated in these cases. The procedure is also proscribed in nonfasting patients and in those with severe bleeding diathesis.

Although transesophageal and transthoracic imaging are based on the principles of ultrasonography, transesophageal echocardiography is superior in most cases. The retrocardiac approach produces higher resolution images, avoids tissues, improves ultrasound transmission, and avoids the necessity of interrupting a thoracic procedure for imaging. Finally, the location affords excellent visualization of both valvular prostheses and aortic anatomy. In fact, because it does not require a contrast medium and can be done at the bedside, transesophageal echocardiography may be preferable to computed tomography and angiography for evaluating aortic dissection in centers with adequate facilities and experienced staffs.

In the past several years, anesthesiologic and critical care uses for transesophageal echocardiography have expanded rapidly. Preoperatively, it is useful for evaluating baseline cardiac performance and valvular anatomy and can define intracardiac vegetations, masses, and thrombi with exquisite detail. Postoperatively, it can complement other monitoring methods in the intensive care unit to follow hemodynamics and cardiac function.

Intraoperative transesophageal imaging is now routinely done during cardiac and high-risk operations in many centers. Transesophageal echocardiography can detect myocardial ischemia at its earliest observable stage, when ventricular relaxation becomes impaired and regional wall motion abnormalities occur. These changes are seen before electrocardiographic ST segment shifts and provide the opportunity to promptly recognize and treat intraoperative ischemia. In addition, pulsed-wave Doppler examination of pulmonary venous flow may help predict changes in pulmonary capillary wedge pressure and associated volume shifts. Finally, transesophageal echocardiography is often invaluable for the intraoperative assessment of valvular insufficiency, repair, and replacement. All of these benefits depend on physicians' experience and procedural skill. Many transesophageal echocardiography determinants are semiquantitative in nature, and thorough training in echocardiography is essential to avoid misinterpretations.

Several recent advances are important to note. Biplanar transesophageal echocardiography probes with two orthogonally arranged crystal arrays were introduced several years ago and allow both transverse and longitudinal cross-sectional imaging of the heart. In the near future, multiplanar probes with electronically adjustable transducer angulation will become generally available. These will permit visualizing almost any imaging plane and will compensate for a lack of probe flexibility within the esophagus.

Echocardiographic contrast material for intravascular and intracardiac administration is now being commercially produced. This fluid is extremely "echo-dense" and reflects ultrasound waves efficiently, even at relatively low concentrations. Echocardiographic contrast can be administered to the right or left ventricle to enhance endocardial border definition and can also be infused intraoperatively into coronary arteries or bypass grafts. In the latter cases, the contrast is carried into the myocardium, which then becomes noticeably brighter on echocardiographic imaging. This is a promising new technique to assess the patency of coronary bypass grafts before the operation is completed and the chest is closed.

A development called acoustic quantification is also now available. With this technology, endocardial borders in a region of interest are automatically defined and enhanced, and real-time changes in left or right ventricular cross-sectional areas can be instantaneously calculated and displayed. With advanced instruments, ultrasonic tissue characteristics of the myocardium can also be studied. This may prove useful in assessing cardiac performance, intrinsic myocardial abnormalities, and, indirectly, ventricular stroke volumes. The combination of multiplanar imaging and acoustic quantification could ultimately lead to clinically practical three-dimensional imaging of the heart.

Timothy L. Feng
Daniel G. Blanchard, M.D.
San Diego, California

REFERENCES

Cahalan MK: Intraoperative monitoring, chap 10, *In* Dittrich HC (Ed): Clinical Transesophageal Echocardiography. St Louis, Mo, Mosby-Year Book, 1992, pp 141-8.

Lee E, Schiller NB: Ambulatory and intraoperative transesophageal echocardiography. *Cardiol Clin* 1989; 7:511-24.

Matsuzaki M, Toma Y, Kusukawa R: Clinical applications of transesophageal echocardiography. *Circulation* 1990; 82:709-22.

New Applications of Propofol

SINCE its introduction in 1989, propofol has rapidly become popular for inducing and maintaining anesthesia. Propofol has two unusual characteristics that account for this popularity. On the pharmacokinetic side, propofol has a rapid clearance, 1.5 to 2 liters per minute, which exceeds hepatic blood flow. Propofol is also highly fat soluble and sequesters in fat following long infusions. These pharmacokinetic properties result in a rapid decrease in propofol concentration following continuous infusions, regardless of the infusion duration. On the pharmacodynamic side, subhypnotic doses of propofol do not cloud consciousness. The net effect is that patients recover rapidly at the end of long infusions.

The U.S. Food and Drug Administration this year approved the use of propofol for "monitored anesthesia care sedation." In this situation, propofol, when compared with midazolam hydrochloride, allows a more rapid titration to the level of sedation and a faster return of clearheadedness. The more rapid blood-brain equilibration of propofol (one to two minutes) compared with midazolam (two to four minutes) means that the level of consciousness can be more rapidly decreased with propofol in anticipation of an imminent noxious stimulus. Once the stimulus is over, the patient can be returned to a lighter level of sedation more rapidly with propofol than with midazolam. This is particularly important if the patient must occasionally interact with the surgeon. Older patients sometimes become excited with the use of midazolam, but not with propofol. Like midazolam, propofol is a potent ventilatory depressant, particularly when combined with opioids. Therefore, the use of propofol for sedation is recommended only when administered by anesthesiologists or other personnel who have been trained in acute airway management.

Propofol is also being investigated for the long-term sedation of intubated patients in intensive care units. The current standard in many hospitals is a continuous infusion of midazolam and an opioid. This regimen results in a substantial accumulation of midazolam and prolonged sedation when the infusion is terminated. The high clearance of propofol permits a rapid decrease in propofol concentrations, even after infusions of one to two weeks. This allows for awakening a patient at regular intervals for the frequent assessment of neurologic status. Provided that the patient has adequate cardiac filling pressures and that judicious titration is used to achieve the appropriate level of sedation, propofol used for sedation in the intensive care unit is usually not associated with the hypotension that accompanies the induction of anesthesia in an operating room. Additional benefits of using propofol in critically ill patients are a decreased metabolic oxygen requirement, with a concomitant increase in mixed venous oxygen saturation, and little need for vasodilators or negative inotropes in hyperdynamic patients. As propofol has no intrinsic analgesic properties, it is helpful to give a small amount of an opioid concurrently. This reduces the amount of propofol required to produce the desired level of sedation.

Propofol also appears to have antiemetic properties. Many of the initial clinical studies documented a low incidence of nausea and vomiting following propofol anesthesia. In several recent studies, propofol was found to be effective in treating chemotherapy-induced emesis. The propofol concentrations that appear effective in treating nausea are well below those associated with sedation.

Steven L. Shafer, M.D.
Palo Alto, California

REFERENCES

Borgeat A, Wilder-Smith OH, Saiah M, Rifat K: Subhypnotic doses of propofol possess direct antiemetic properties. *Anesth Analg* 1992; 74:539-41.

Shafer A, Doze VA, White PF: Pharmacokinetic variability of midazolam infusions in critically ill patients. *Crit Care Med* 1990; 18:1039-41.

Shelly MP, Sultan MA, Bodenham A, Park GR: Midazolam infusions in critically ill patients. *Eur J Anaesthesiol* 1991; 8:21-7.

Sher C, McDowall R, Barst S: Propofol for the prevention of chemotherapy induced nausea and vomiting in pediatric cancer patients (Abstr). *Anesthesiology* 1991; 75:A1050.

'Do-Not-Resuscitate' Orders in Operating Rooms

PATIENTS with do-not-resuscitate (DNR) orders may come to operating rooms for procedures to palliate symptoms. They also may need operations to treat lesions related or unrelated to the disease that led to the DNR decision. How is the DNR status to be handled by operating room personnel? This is an important question because common interventions in operating rooms could be labeled resuscitation in other settings. It may be difficult to separate resuscitation required to reverse acute anesthesia- or surgery-related problems from that needed to reverse events caused by the underlying disease.

At least four options exist for dealing with DNR orders perioperatively:

- Allow the preoperative DNR order to stand, but suspend the DNR order during and soon after the operative period. In this instance DNR plans can be reinstituted when the patient has recovered from the acute effects of anesthesia;

- Life-sustaining measures instituted intraoperatively can be withdrawn postoperatively. For example, if a patient has become ventilator dependent, an agreement to discontinue such treatment can be documented preoperatively;

- Provide all resuscitative measures short of cardiac massage;

- Suspend DNR orders but reinstate them intraoperatively if cardiac arrest occurs related to the disease that led to the DNR orders, such as hypovolemic shock during a portacaval shunt in a patient with advanced cirrhosis and encephalopathy.

In recent lively and thoughtful debates about DNR orders in patients requiring surgical treatment, each of the above options or blends of these options has been proposed. Federal law (Patient Self-Determination Act of 1990) requires that patients entering institutions receiving Medicare or Medicaid funds must be advised of their right to issue advanced directives. All hospitals seeking accreditation from the Joint Commission on Accreditation of Healthcare Organizations must have DNR policies. Policies should include the management of DNR orders when surgery and anesthesia are required.

Communication among surgical professionals, the primary care team, and the patient and family about DNR orders in the perioperative period must be clear and open. The goal is to remove ambiguity about the perioperative care of patients to the greatest possible extent. This will require patience and time. Both must be provided.

Wendell C. Stevens, M.D.
Portland, Oregon

REFERENCES

Cohen CB, Cohen PJ: Do-not-resuscitate orders in the operating room. *N Engl J Med* 1991; 325:1879-82.

Martin RL, Soifer BE, Stevens WC: Ethical issues in anesthesia: Management of the do-not-resuscitate patient. *Anesth Analg* 1991; 73:221-5.

Walker RM: DNR in the OR: Resuscitation as an operative risk. *JAMA* 1991; 266:2407-12.

Younger SJ, Cascorbi HF, Shuck IM: DNR in the operating room: Not really a paradox. *JAMA* 1991; 266:2433-4.

Novel Approaches to the Treatment of Neuropathic Pain

INJURY or disease of the central or peripheral nervous system causes some of the most agonizing and intractable chronic pain problems. Pain can result from nervous system injury caused by direct trauma (brachial plexus avulsion, phantom limb pain), ischemia (thalamic syndrome), infection (tabes dorsalis, postherpetic neuralgia), metabolic derangements (diabetic neuropathy), or tumor invasion (lumbosacral plexopathy). Patients with these syndromes may describe sensations of intolerable burning, searing, crushing, electric shocks, pins and needles, or tearing of the flesh and often suffer serious disability as a result. Suicide is an unusual but well-described complication of neuropathic pain.

Anesthesiologists are frequently requested to consult on patients with neuropathic pain with the hope that a patient may respond favorably to a neural blockade technique. Unfortunately, even when there is an initial favorable response to a block, the results may not be long-lasting, and medical management often assumes an important role. The treatment of neuropathic pain is complicated by the fact that routine analgesics such as nonsteroidal anti-inflammatory drugs and opiates may be ineffective. In fact, a lack of pain relief from standard doses of opiates may point to the diagnosis of neuropathic pain. Fortunately other types of drugs not normally classified as analgesics are often effective for the relief of neuropathic pain. These include tricyclic antidepressants, anticonvulsants, parenteral administration of topical anesthetics, and α-adrenergic antagonists.

It is now well established that tricyclic antidepressants relieve pain in a variety of neuropathic conditions. This analgesic action of the tricyclics is independent of any antidepressant effect. Amitriptyline hydrochloride has been the most extensively studied and most widely used tricyclic for the relief of neuropathic pain, but many other tricyclics have proved to be effective, including nortriptyline hydrochloride, imipramine hydrochloride, and desipramine hydrochloride. Toxicity is rarely a problem in the dose ranges normally required for analgesia, and tricyclics do not produce euphoria or tolerance so that abuse potential is low. The mechanism of analgesia from the tricyclic compounds is thought to be an inhibition of the uptake of biogenic amines, such as serotonin and norepinephrine, which are known to inhibit pain-transmitting neurons.

Anticonvulsant drugs may also be useful for the treatment of neuropathic pain, particularly when a patient reports a paroxysmal lancinating or "electric shock" component to the pain. Experimental evidence suggests that injured nerves may develop hyperexcitability and spontaneous ectopic activity. Thus, paroxysmal neuralgic pain resembles epilepsy in certain respects, and in these patients antiepileptic drugs may provide pain relief. Carbamazepine is now the treatment of choice for trigeminal neuralgia, and more recently clonazepam and sodium valproate have been shown to be useful for the treatment of various lancinating pains, as may occur in postherpetic neuralgia, tabes dorsalis, phantom limb pain, diabetic neuropathy, and the thalamic syndrome.

The intravenous infusion of lidocaine has been shown to suppress spontaneous activity in neuromas and to reduce pain in diabetic neuropathy and other neuropathic conditions. Intravenous lidocaine infusion may eventually prove to be a useful test to identify a neuropathic component to a chronic pain syndrome. These observations have stimulated interest in the use of the oral antiarrhythmic and lidocaine congener mexiletine hydrochloride, which was recently shown to reduce neuropathic pain in patients in whom alternative medical regimens had been unsuccessful. In particular, patients with a "burning" component to the pain seemed to benefit from oral mexiletine, and side effects were minimal. The mechanism of action of these drugs is unclear, but may involve alterations in spinal cord processing, rather than by peripheral axonal or receptor blockade.

Neuropathic pains are often maintained or augmented by efferent activity in the sympathetic nervous system, as occurs in the reflex sympathetic dystrophy syndrome. This condition has traditionally been diagnosed and treated by repeated regional anesthetic blocks of the appropriate sympathetic ganglia. Recent experimental evidence has suggested that sympathetically maintained pains are mediated largely through α-adrenergic mechanisms. The intravenous infusion of phentolamine mesylate has been described as a reliable, specific, and easy-to-do diagnostic test for sympathetically maintained pain. For patients who respond to phentolamine infusion, daily treatment with oral phenoxybenzamine hydrochloride or other α-adrenergic antagonists may permanently eradicate sympathetically mediated components of the pain. Side effects such as orthostatic hypotension may limit therapy, however.

Robert W. Presley, M.D.
Los Gatos, California

REFERENCES

Arner S: Intravenous phentolamine test: Diagnostic and prognostic use in reflex sympathetic dystrophy. *Pain* 1991; 46:17-22.

Chabal C, Jacobson J, Mariano A, et al: The use of oral mexiletine for the treatment of pain after peripheral nerve injury. *Anesthesiology* 1992; 76:513-7.

Postoperative Pain

THE side effects of postoperative pain management have long been a focus of attention. Concern has now shifted to the possible morbidity of inadequate therapy; indeed, there is a growing body of evidence that postoperative pain may have harmful physiologic and psychological effects. Specifically, pain may adversely affect respiratory function, contribute to the development of a stress response, increase cardiac morbidity, and prolong hospital stays.

New analgesic agents have led to improvements in acute pain management. The recent approval of ketorolac tromethamine, a potent nonsteroidal anti-inflammatory agent, has provided physicians with a nonopioid alternative for the treatment of mild to moderate pain and a useful adjunct to decrease the opiate requirement of moderate to severe pain. Possible advantages include the lack of sedation, respiratory depression, or disturbance of bowel and bladder function. Unfortunately, the use of ketorolac may be associated with serious side effects, including gastrointestinal ulceration, platelet dysfunction, and nephrotoxicity. Patients who are hypovolemic or have preexisting renal insufficiency may be particularly vulnerable to the nephrotoxicity.

Even more important than the introduction of new analgesic agents has been the increased use of regional anesthetic techniques and the development of better methods of drug delivery, particularly patient-controlled analgesia and the spinal administration of narcotics. Although narcotics (fentanyl citrate) may also be administered transdermally, this route is not well suited for the initial treatment of acute postoperative pain because 12 to 24 hours are required to reach a steady state. Transdermal administration, however, appears to have a useful role in the treatment of patients who, after several days, still cannot take anything orally and have relatively stable pain.

Numerous surveys have documented that scheduled or patient-requested, intermittent, intramuscular administration of narcotics fails to control postoperative pain. Patient-controlled analgesia effectively circumvents problems of variable absorption and delays. When in pain, the patient depresses a button, causing the delivery of a fixed intravenous dose from an infusion pump. The physician sets both the dose of drug delivered by the machine and the "lockout" interval, which is the time after a dose during which a second dose cannot be delivered by the machine. The "lockout" should be of sufficient duration to allow the patient to obtain near-peak effect from the dose already received, thereby safeguarding against overdosage due to cumulative effects. Along with administration based on patient demand, a low-dose continuous or basal infusion can be provided. The theory is that supplementation with a basal infusion should provide more effective pain control and encourage uninterrupted sleep; unfortunately, although the data are somewhat equivocal, they do not appear to confirm these benefits. In addition, the inclusion of a basal infusion may decrease the relative safety of the technique. Most studies document improved control of pain with patient-controlled analgesia. Moreover, patient acceptance is exceptionally high; in fact, in one controlled study, the use of patient-controlled analgesia resulted in greater patient satisfaction than the epidural administration of morphine sulfate, even though patients receiving epidural morphine had better analgesia.

Unlike the administration of systemic narcotics, the spinal administration of opiates can produce profound analgesia without a significant depression of the sensorium. Selective spinal analgesia can be achieved using either epidural or intrathecal opioid administration, but the use of an epidural catheter technique permits continuous or repeated injections to provide analgesia for prolonged periods and allows for the substitution or addition of local anesthetics. Although clinical management is more complex when local anesthetics are combined with spinal opioids, the combination can increase analgesia, delay tachyphylaxis, and reduce the side effects that occur when either technique is used alone.

The importance of aggressive postoperative pain management has been underscored by the recent publication of clinical practice guidelines for acute pain management by the Agency for Health Care Policy and Research. The guidelines stress a collaborative interdisciplinary approach; an individualized plan developed preoperatively; assessment and frequent reassessment of the patient's pain; the use of both drug and nondrug therapies; and a formal institutional approach, including incorporating a quality assurance program.

The effective control of postoperative pain is important and necessary not only for patient comfort but also for rapid recovery and optimal results. The effectiveness of newer methods such as patient-controlled analgesia and intraspinal techniques and the availability of newer, more potent, nonsteroidal anti-inflammatory drugs permit far more effective management of postoperative pain than with traditional intramuscular therapy.

Kenneth Drasner, M.D.
San Francisco, California

REFERENCES

Acute Pain Management Guideline Panel: Acute Pain Management: Operative or Medical Procedures and Trauma—Clinical Practice Guideline, AHCPR publication No. 92-0032. Rockville, Md, Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services, February 1992.

Drasner K, Katz JA, Schapera A: Control of pain and anxiety, chap 82, *In* Hall JB, Schmidt GA, Wood LDH (Eds): Principles of Critical Care. New York, NY, McGrawHill, 1992, pp 958-73.

Perioperative Myocardial Ischemia

DESPITE recent advances in the diagnosis and treatment of coronary artery disease, the incidence of perioperative cardiac morbidity remains high. As a result, considerable research has been devoted to investigating perioperative myocardial ischemia as a possible reversible predictor of adverse surgical consequences.

Intraoperative electrocardiographic changes consistent with myocardial ischemia occur in 18% to 74% of patients with coronary artery disease who are having noncardiac operations. A recent study shows that ST segment changes occur laterally (V4, V5) and have a variable duration. Although intraoperative ischemia can be precipitated by increases in myocardial oxygen demand due to tachycardia and hypertension, 50% or more of the ischemic episodes may be unrelated to increases in indices of oxygen demand, suggesting that a primary decrease in the oxygen supply may be an important cause.

Although a previous study suggested that the occurrence of ischemia in the pre-bypass period in patients undergoing coronary artery bypass grafting may increase the risk of postoperative myocardial infarction, more recent studies show that a chronic pattern of ischemia exists preoperatively in patients having coronary artery bypass grafting as well as those having noncardiac surgical procedures. Furthermore, the intraoperative pattern of ischemia is no worse than the preoperative pattern, implying that anesthesia and surgery may not be as stressful as previously assumed and that the intraoperative pattern may simply recapitulate the ongoing preoperative pattern.

Finally, it is now established that postoperative ischemia is even more prevalent than ischemia occurring before or during an operation and is the most important predictor of adverse cardiac events in high-risk patients undergoing noncardiac operations. In fact, postoperative myocardial ischemia as detected by Holter electrocardiographic monitoring confers a 9.2-fold increase in the odds of a cardiac event. No other clinical, historical, or perioperative variable was associated independently with adverse cardiac results, including the cardiac risk index, a history of previous myocardial infarction or congestive heart failure, or the occurrence of ischemia before or during an operation. Accordingly, increased attention and resources should be focused on the postoperative period. Clinical trials of perioperative antiischemic therapies are currently under way. The results from these trials will determine whether the incidence of perioperative cardiac morbidity can be reduced by the prevention of postoperative ischemia.

Jacqueline M. Leung, M.D.
San Francisco, California

REFERENCES

Knight AA, Hollenberg M, London MJ, et al: Perioperative myocardial ischemia of the preoperative ischemic pattern. *Anesthesiology* 1988; 68:681-8.

London MJ, Hollenberg M, Wong MG, et al: Intraoperative myocardial ischemia Localization by continuous 12-lead electrocardiography. *Anesthesiology* 1988; 69:232-41.

Mangano DT: Perioperative cardiac morbidity. *Anesthesiology* 1990; 72:153-84.

Mangano DT, Browner WS, Hollenberg M, London MJ, Tuban JF, Tateo IM: Association of perioperative myocardial ischemia with cardiac morbidity and mortality in men undergoing noncardiac surgery. *N Engl J Med* 1990; 323:1781-8.

Sedating Patients in Intensive Care Units

ANXIETY, sleep deprivation, and pain are extremely common problems in patients in intensive care units and can lead to delusions, delirium, or psychosis. The neuroendocrine stress responses often associated with noxious conditions can produce deleterious hemodynamic, metabolic, nutritional, and immunologic changes. Inadequate control of any of these problems frequently results in unnecessarily large doses of sedatives. Coughing with an endotracheal tube and fighting the ventilator complicate the management of patients on ventilatory support. Asynchrony between spontaneous ventilatory efforts and mechanical breaths predisposes patients to pulmonary barotrauma, interferes with alveolar gas exchange, and increases the work of breathing. Neuromuscular paralysis for the control of inappropriate (central) hyperventilation mandates that sedation also provide amnesia.

Older sedation practices relied primarily on intermittent intravenous doses of morphine sulfate or meperidine hydrochloride. Diazepam or pentobarbital was given when amnesia or hypnosis was required. Unfortunately, boluses of these long-acting agents often caused serious circulatory and respiratory depression, and the high doses of opiates decreased gastrointestinal motility and prolonged ileus.

Midazolam hydrochloride, a short-acting benzodiazepine, has a pharmacokinetic profile that readily allows titration to effect when given by continuous intravenous infusion. Loading is best achieved by 0.5-mg increments followed by an infusion of 0.015 to 0.2 mg per kg per hour. Respiratory or circulatory depression is generally minimal, and even after prolonged infusions its effects usually dissipate within two hours after the infusion is stopped. The latter facilitates a rapid weaning from mechanical ventilation and neurologic evaluation. Withdrawal symptoms are uncommon, but tolerance may develop in some patients.

Supplementing midazolam is important whenever a patient has substantial pain, is on a ventilator, or tolerance develops. A concomitant infusion of an opioid or ketamine hydrochloride augments the sedation, provides analgesia, and reduces drug requirements. Adding fentanyl citrate, 0.5 to 2 µg per kg per hour, sufentanil citrate, 0.05 to 0.2 µg per kg per hour, or alfentanil hydrochloride, 5 to 20µg per kg per hour, can produce excellent analgesia and reduces midazolam requirements. Alternatively, ketamine, 0.15 to 0.5 mg per kg per hour, can also provide powerful analgesia. Unlike opioids, ketamine is a bronchodilator and mild respiratory stimulant. Psychotomimetic reactions, such as hallucination and nightmares, are uncommon when ketamine is combined with a benzodiazepine.

Propofol, a short-acting intravenous anesthetic with a rapid onset and termination, is an excellent sedative at low doses—I to 6 mg per kg per hour. Dose-dependent respiratory depression and hypotension can be observed after administering a bolus but are generally minimal when propofol is given by infusion at this dose range. Propofol's principal advantage is that its effects usually dissipate within 15 to 20 minutes after the infusion is stopped. Withdrawal symptoms have been reported following prolonged propofol infusion.

Maged S. Mikhail, M.D.
Duraiyah Thangathurai, M.D.
Los Angeles, California

REFERENCES

Aitkenhead AR: Analgesia and sedation in intensive care. *Br J Anaesth* 1989; 63:196-206.

Harris CE, Grounds RM, Murray AM, Lumley J, Royston D, Morgan M: Propofol for long term sedation in the intensive care unit: A comparison with papaveretum and midazolam. *Anaesthesia* 1990; 45:366-72.

Tobias JD, Martin LD, Wetzel RC: Ketamine by continuous infusion for sedation in the pediatric intensive care unit. *Crit Care Med* 1990; 18:819-21.

Perioperative Temperature Control

HUMANS normally maintain a core body temperature near 37°C. This temperature is maintained because deviations of only a few tenths of a degree trigger thermoregulatory responses, including vasoconstriction, shivering, active vasodilation, and sweating. General anesthetics profoundly impair normal thermoregulatory processing. For example, the core temperature triggering vasoconstriction is decreased from about 37°C to about 34°C by halothane, nitrous oxide and fentanyl citrate, nitrous oxide and propofol, and isoflurane. The exact triggering threshold depends on the anesthetic type and dose, but is similar in infants, children, and adults. In contrast, the core temperature triggering sweating and active vasodilation is increased about a degree by surgical concentrations of isoflurane and enflurane. The interthreshold range (core temperature not triggering thermoregulatory responses) thus is increased by general anesthesia from about 0.6°C to about 4°C. Most patients become hypothermic during surgical procedures, and hypothermia usually develops in three distinct phases. During the first hour, the core temperature decreases about 1 °C. Surprisingly, undressing patients in a cold room and washing their skin with fluid that is subsequently allowed to evaporate contributes little to the hypothermia. Anesthetic-induced vasodilation only minimally increases heat loss from the skin surface. Similarly, inducing anesthesia only minimally reduces metabolic heat production. Core hypothermia actually results because vasodilation allows heat in warm core tissues to escape into the cooler peripheral thermal compartment. This reduces the core temperature, increases the peripheral temperature, and leaves the body heat content unchanged.

Redistribution hypothermia is followed by a slow linear decrease in the core temperature, lasting two to three hours. This linear decrease apparently results simply from heat loss exceeding metabolic heat production.

Finally, after three to four hours of anesthesia, the core temperature becomes constant and does not decrease further even during prolonged surgery.

Intraoperative hypothermia is most common in infants and children because of a high surface area-to-mass ratio and in elderly patients because thermoregulatory responses are impaired. Mild hypothermia—2°C to 3°C below normal— protects against hypoxia, ischemia, and malignant hyperthermia. Consequently, hypothermia is appropriate in patients in whom ischemia can be expected, such as during carotid and neurosurgical procedures. Conversely, hypothermia decreases drug metabolism, impairs coagulation, decreases resistance to wound infections, and provokes postanesthetic shivering. Most patients should therefore be maintained at normal temperature. Hypothermia is best treated using a high ambient temperature or forced-air warming. Circulating-water warmers placed over patients substantially decrease heat loss but are relatively ineffective when positioned under patients. Warming intravenous fluids is critical when large volumes are administered. Airway heating and humidification transfer little heat and are not worth the expense in adult patients.

Daniel I. Sessler, M.D.
San Francisco, California

REFERENCES

Hynson JM, Sessler DI: Prevention and treatment of intraoperative hypothermia. *J Clin Anesth* 1992; 4:194-9.

Just B, Delva E, Camus Y, Lienhart A: Oxygen uptake during recovery following naloxone. *Anesthesiology* 1992; 76:60-4.

Sessler DI, McGuire J, Moayeri A, Hynson JM: Isoflurane-induced vasodilation minimally increases cutaneous heat loss. *Anesthesiology* 1991; 74:226-32.

Postoperative Apnea Syndrome in Premature Infants

THE predisposition of preterm infants to episodes of apnea in the postoperative period has been recognized for a decade. What progress has been achieved during this time in our understanding of the etiology of this problem, and how should these patients be managed?

Infants younger than 45 weeks of conceptual age (conceptual age is the sum of gestational age and postnatal age) are more likely to be affected. Although conceptual age is a significant predictor of problems, there is also a suggestion that infants who were born at an early gestational age are at a further increased risk. In addition, a number of other predisposing factors have become recognized. These include anemia, a history of the respiratory distress syndrome, persistent chronic respiratory disease, and previous apneic episodes. Although a history of apnea is clearly important, infants with no previous episodes of apnea may also be at risk to suffer an apneic episode in the postoperative period. Apnea usually occurs within the first few hours after the operation, but in smaller infants may occur much later. The apnea is central in origin in most instances, but obstructive apnea may also occur. Apnea is more likely to occur after general anesthesia, but life-threatening apnea may rarely occur even if only regional analgesia has been used.

Current recommendations are that the management of preterm infants generally should include hospital admission and careful postoperative monitoring of all surgical patients who are younger than 50 weeks of postconceptual age. Such infants should be treated in hospitals that have a neonatal service and a neonatal intensive care unit. After the operation they should be monitored with an apnea alarm in an area under close nursing supervision for 12 to 24 hours or more, depending on their history. The presence of anemia, chronic respiratory disease, or persistent episodes of apnea are indications to admit older infants. The use of spinal or caudal anesthesia may be advantageous, especially in those infants with a previous history of prolonged ventilatory support.

Intravenous caffeine therapy, 10 mg per kg, given at the induction of anesthesia has been suggested to reduce the danger of postoperative apnea, but close postoperative observation is still recommended. At present, the use of caffeine therapy is not common. The influence of theophylline therapy on the danger of postoperative apnea has not been fully studied.

David I. Steward, M.B.
Los Angeles, California

REFERENCES

Cox RG, Goresky GV: Life-threatening apnea following spinal anesthesia in former premature infants. *Anesthesiology* 1990; 73: 345-7.

Gunter JB, Watcha MF, Forestner JE, et al: Caudal epidural anesthesia in conscious premature and high-risk infants. *J Pediatr Surg* 1991; 26:9-14.

Kurth CD, LeBard SE: Association of postoperative apnea, airway obstruction, and hypoxemia in former premature infants. *Anesthesiology* 1991; 75:22-6.

Welborn LG, Hannallah RS, Luban NL, Fink R, Ruttimann UE: Anemia and postoperative apnea in former preterm infants. *Anesthesiology* 1991; 74:1003-6.

Autologous Blood Transfusion Therapy

THE probability that homologous blood transfusions will transmit infectious diseases has decreased with thorough blood donor screening and sophisticated laboratory testing. The risk of contracting a potentially lethal infection nonetheless remains finite (one in about 40,000 units for the human immunodeficiency virus [HIV]). Autologous blood transfusion is the safest alternative for some patients having surgical procedures. In California, whenever there is a reasonable possibility that a patient having a procedure will require a blood transfusion, the patient must be informed of the risks and benefits of the various options for managing transfusion therapy.

Few surgical procedures are commonly associated with a critical volume of blood loss that warrants transfusion therapy. Patient-specific factors such as size, cardiovascular status, pulmonary function, and hematologic profile are important in decisions relating to transfusion. Autologous blood for transfusion in the perioperative period may be obtained by preoperative autologous donation, normovolemic hemodilution, intraoperative blood salvage, or postoperative blood salvage. Alone or in combination, these can decrease or eliminate the need for homologous blood transfusion.

As many as four to five autologous units can be safely donated preoperatively during the six-week shelf life of erythrocytes collected in adenine-supplemented preservatives. The plasma can be freshly frozen. The hematocrits of autologous donors are monitored, and treatment with oral iron is routine. The value and role of recombinant human erythropoietin remain under investigation. Because the morbidity of donation is predominantly related to vasovagal reactions, clinically adverse hemodynamic sequelae are more likely in patients with cardiac or cerebrovascular disease. Older children and elderly patients can qualify, but Jehovah's Witnesses will not accept this procedure. Autologous units are tested for HIV, hepatitis B and C, syphilis, and the human T-cell lymphotropic virus type I. Each blood bank disposes autologous units that test positive according to their own policies. Complications can occur when autologous units are transfused, usually the result of clerical or laboratory errors or circulatory overload in the patient.

Normovolemic hemodilution is the withdrawal of a patient's whole blood and replacement with a larger volume of intravenous crystalloid—or an equal volume of colloid— which is done after inducing anesthesia and before beginning the operation. Hemodiluted patients lose fewer erythrocytes per volume of surgically shed blood. The anticoagulated withdrawn blood may be stored at room temperature for a maximum of six hours before being reinfused. Normovolemic hemodilution is contraindicated in patients in whom the compensatory increase in cardiac output is physiologically impossible or harmful.

Intraoperative blood salvage is carried out for noncontaminated, nononcologic surgical procedures that are characterized by the loss of substantial quantities of blood. Jehovah's Witnesses may be receptive to either normovolemic hemodilution or intraoperative blood salvage. The most frequently used technology for this procedure incorporates canister collection arrangements, centrifuge-based erythrocyte salvage contrivances, and supplemental washing devices. The capacity and speed of this equipment can provide almost 3 liters of saline-suspended packed cells per hour. Salvaged erythrocytes have survival times equivalent to those of homologous erythrocytes, but because intraoperative blood salvage fails to save functional platelets and clotting proteins, a dilutional coagulopathy is more likely with an increased volume of blood salvaged. Washing also removes anticoagulant used for collection and noninhalational anesthetic drugs. The potential for air embolism mandates using safeguards. Published experience now supports cautiously expanding the use of intraoperative blood salvage to selected patients having extensive cancer surgery (reducing the immunosuppressive properties of homologous transfusion); emergency traumatically contaminated body cavities (mandating a washing technique and antibiotic therapy); ectopic pregnancy; and cesarean section deliveries in which blood salvage is initiated after completing a separate and discarded suctioning of amniotic fluid from the surgical field. Unprocessed (unwashed) intraoperative blood salvage results in transfusing debris and free hemoglobin from the operative field, both of which have been implicated in cases of disseminated intravascular coagulation and organotoxicity. An absolute contraindication to the procedure is the surgical use of a microfibrillar collagen hemostat. Deliberate hypotensive anesthesia in appropriately selected patients will decrease the volume of blood obtained.

The development of clinically safe and effective oxygen carriers such as cell-free hemoglobin-based solutions and perfluorocarbon emulsions will alleviate some of the demand for blood replacement therapy

Stephen H. Jackson, M.D.
Monte Sereno, California

REFERENCES

Spiess BD, Sassetti R, McCarthy RJ, Narbone RF, Tuman KJ, Ivankovich AD: Autologous blood donation: Hemodynamics in a high-risk patient population. *Transfusion* 1992; 32:17-22.

Tasaki T, Ohto H, Hashimoto C, Abe R, Saitoh A, Kikuch S: Recombinant human erythropoietin for autologous blood donation: Effects on perioperative red-blood-cell and serum erythropoietin production. *Lancet* 1992; 339:773-5.

Williamson KR, Taswell HF: Intraoperative blood salvage: A review. *Transfusion* 1991; 31:662-75.

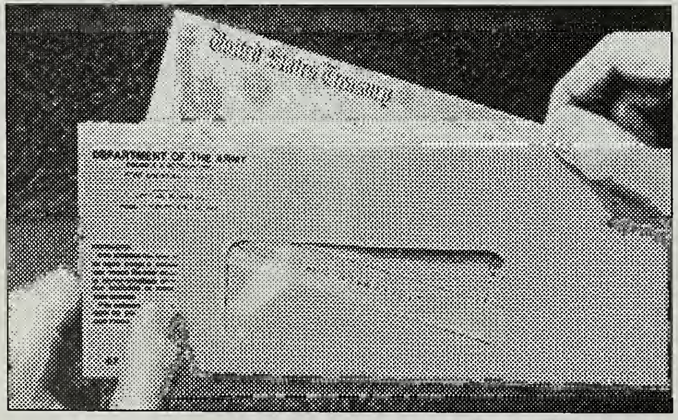
ADVISORY PANEL TO THE SECTION ON ANESTHESIOLOGY

Stephen H. Jackson, M.D.
Advisory Panel Chair
CMA Council on Scientific Affairs Representative
Monte Sereno

Thomas Cromwell, M.D.
CMA Section Chair
Belvedere
Jack L. Moore, M.D.
CMA Section Secretary
Los Angeles
Thomas W. Feeley, M.D.
CMA Section Assistant Secretary
Partola Valley
C. Philip Larson, Jr, M.D.
Immediate Past Panel Chair
Stanford
Floyd S. Brauer, M.D.
Loma Linda University
Frank H. Sarnquist, M.D.
Stanford University
John H. Eisele, Jr, M.D.
University of California
Davis
Steven J. Barker, M.D.
University of California
Irvine
Joseph C. Gabel, M.D.
University of California
Los Angeles
Harvey M. Shapiro, M.D.
University of California
San Diego
Ronald D. Miller, M.D.
University of California
San Francisco
G. Edward Morgan, M.D.
Section Editor
University of Southern California
Richard E. Tirrell, M.D.
California Society of Anesthesiologists
Moraga
Patricia A. Kapur, M.D.
California Society of Anesthesiologists
Northridge
Bernard D. Morgan, M.D.
American Society of Anesthesiologists
Riverside
Timothy Feng
Medical Student Representative
University of California
San Diego

ANESTHESIOLOGISTS AND SURGEONS: COULD YOU USE AN EXTRA $9,000?

If you're a resident in anesthesiology or surgery, the Army Reserve will pay you a yearly stipend which could total in excess of $9,000 in the Army Reserve's Specialized Training Assistance Program (STRAP).



You will have opportunities to continue your education and attend conferences, and we will be flexible about scheduling the time you serve. Your immediate commitment could be as little as two weeks a year, with a small added obligation later on.

Get a maximum amount of money for a minimum amount of service. Find out more by contacting an Army Reserve Medical Counselor.

CALL COLLECT (203) 624-5325

ARMY RESERVE MEDICINE. BE ALL YOU CAN BE.®

The Connecticut State Medical Society and Malpractice
The First 180 Years

W. RAYMOND JAMES, M.D.

IN May 1847, at the annual meeting of the Connecticut State Medical Society (CSMS), Dr. Theodore Sill gave a noteworthy dissertation on the treatment of typhus fever. It was remarkable that Dr. Sill decried the then current methods of treatment. Bleeding, blistering, etc., he said, were "absolutely contraindicated." He considered laxatives and emetics to be harmful and felt they could induce the terminal stages of this frequently fatal disease. He also opposed fasting and other dietary fads, recommending instead broth made from meat extracts, "as much as the patient could take," and "stimulants," preferably opium and possibly alcohol, "if well diluted," to control delirium and extreme discomfort. Claiming that his methods(1992 physicians could hardly have provided *better* care with the tools of that time!) were successful he nevertheless "feared the accusation of malpractice" in case of a death.

Dr. Sills's article was also remarkable in that in a review of the *Proceedings of the Connecticut State Medical Society* from 1792 to 1891 (99 years!), including all the minutes, the addresses of the presidents and guests, the dissertations (usually one or two doctors were assigned to give a dissertation at each annual or semiannual meeting), county reports, etc., this is the *only* reference to malpractice or medical liability. In spite of the paucity of reference to malpractice it seems reasonable to infer, from Dr. Sills's remarks, malpractice was on the minds of even the best of physicians, and malpractice was usually considered to be a deviation from the usual methods of treatment. Both inferences are supported by Kenneth DeVille in a recent book, *The History of Medical Malpractice During the 19th Century.*[1]

This dearth of reference to malpractice in the society records is surprising in view of the long history of medical liability. Markby's *Elements of Law*[2] reports: "To exact for all injuries both to person and property a payment in money to the person injured appears to have been the first form of legal liability for injuries to private persons alike in *Greece*, in *Rome*, and amongst the *Teutonic Tribes*." It would be interesting to know all that might happen to medical practitioners who were unsuccessful in the ancient civilizations in North Africa, the Near East, the Far East, and the rest of the world in those times.

Most early American medicolegal history dates from early England and English common law. Dr. Andrew Sandor[3] reported that the earliest mention of professional liability occurs in Britton *circa* 1290.[4] In 1553 an early rule was laid down by the English jurist, Sir Anthony Fitzherbert, "It is the duty of every artisan to exercise his art right and truly as he ought."[5] This, and other English common law principles are cited in American trials centuries later.

Dr. Sandor also cited an action in 1374 against one J. Mort, a surgeon, involving treatment of a wounded hand. Because of the "negligence" of the surgeon, "The plaintiff's hand was so impaired that it was maimed to his injury and damage."[6] The surgeon escaped because of a legal technicality but "the rule was clearly laid down that if negligence is proved the law will provide a remedy."

These and other early English cases were referred to as medicolegal or medical liability cases for the word "malpractice" did not appear until 1671.[7] Use of the word "malpractice" was to the physician's advantage in that it suggested injury to a patient (tort law) rather than failure to "perform as he ought" (contract law). Prior to 1969, when it was changed to two years, the Connecticut statute of limitations for tort cases was one year, whereas in contract law the statute of limitation had been at least six years (In about 1930 it was reduced to one year). Apparently the line between tort and contract law is not completely clear even today. Two hundred years ago many cases were tried under contract law to avoid the time limitation on tort cases.

Dr. Sandor extensively reviewed appellate court cases of professional liability in the United States from the first reported case in 1794 to 1955.[8] In the first hundred years only four cases were found in Connecticut and in three of these the plaintiff won. In Connecticut's first malpractice case (also the first appellate court case in the United States), Cross *vs* Guthrie,[9] tried in 1794, Dr. Guthrie was sued by the husband when his wife lived only three hours after a breast operation. It was alleged the doctor operated "in a most unskilled, ignorant and cruel manner," and "... that the defendant had wholly broken and violated his undertaking and promise to the plaintiff to perform the operation skillfully and with safety to his wife." The jury found the defendant guilty and awarded £40 in damages. The case and pleadings followed English common law theory of breach of contract.

Dr. Sandor surveyed only appellate court suits; claims in lesser courts were too numerous, varied, and too frequently unrecorded to make a survey significant. The general opinion seems to be that fewer than 1% of claims tried in lesser courts were appealed, suggesting that there were about 400 claims in Connecticut during the Society's first century. Although this averages only four claims per year, that figure is not impressively low, considering there were so few regular physicians in those years. Quacks or "irregular doctors" abounded in the 19th century, but they were even less subject to suits then than they have been in the 20th century.

Kenneth A. DeVille reported a low incidence of malpractice throughout the country, and presumably in Connecticut, in the early part of the 19th century, with a sharp increase after 1840.[10] He refers to the increase as the "deluge." Beginning violently in New York state, it soon spread to New Hampshire, Vermont, Maine, Massachusetts, then westward to Ohio and on even to San Francisco. Connecticut escaped the "deluge"; the problems of surrounding states were not noted at any time in the Proceedings of the Connecticut State Medical Society. The deluge, at least in part, can be attributed to the following:

- Multiple, weak and competing medical societies. [In contrast, CSMS and the component societies were powerful and well organized.]

- A plethora of poorly educated regular physicians and irregular "doctors" (quacks and cultists). There were fewer then 4,900 physicians in the nation in 1790 and over 40,000 by 1850. The physician to patient ratio had more than doubled, and this did not include the irregulars! [The increase in both real and bogus doctors was far less in Connecticut with only one medical school and none of the diploma mills that had burgeoned in the rest of the north.]

- Medical education had become totally inadequate. The medical school at the University of Pennsylvania (first in this country) opened in 1765 with a four-year curriculum and stiff entrance requirements. By 1789 the curriculum consisted primarily of one year of lectures, and the entrance requirement was the fee to pay the lecturers. DeVille reports that as late as 1870 (the year of beginning revolutionary changes in medical education) perhaps one-half of Harvard's medical students could not write.

- Unprincipled competition, including the urging of even unfounded suits against competing physicians by regular physicians as well as by irregular doctors.

- Jacksonian populism in which the learned were considered to be elitists but were expected to perform as only well-educated physicians could. Even the courts came to look on physicians as tradesmen.

- A slackening in the fatalistic religious views of the colonial churches that all events were determined by divine providence and therefore the deformed leg with imperfect healing was an act of providence and not the fault of the physician. Strong belief in the role of divine providence persisted much longer in the churches in the South than in the North and may explain the low rate of malpractice suits in the South until modern times. The first malpractice suit in Tennessee occurred in 1855. One town in Georgia had no civil suits until 1970!

- Agrarian colonial communities were like large families with the lawyer, physician, teacher, and minister each an important part of the family and expected to do his best but not to be perfect. With industrialization and shifting populations much of the community cohesiveness was lost.

Connecticut escaped most of DeVille's deluge but not the steady increase in malpractice litigation that was to parallel advances in medical care.

There can be little doubt that early physicians were concerned about the quality of medical care. There were efforts by medical societies from the earliest days to increase the knowledge and improve the ability of physicians and the quality of care.

The first application for a state charter for a medical society in Connecticut was made by a group of physicians from Norwich in 1763. The following excerpts from the application indicate their regard for medical education and the qualification of the physician.

> ...the Promoting (of) Medical Knowledge among Physicians is the Necessary and direct means to Restore health and even Preserve Life and is of great Importance as it will Render The Practice of Physic more safe and Serviceable in the Patient and at the same time yeald more Satisfaction and Honour to the Profession....The Physicians in each County for their Edification and Instruction...choose a Committee...to Examine...and Approve...Candidates...for the Practice of Physic.

The charter of the CSMS, in its original form and with early amendments, gave the Society great powers as the licensing agent for physicians in Connecticut. The society was to set standards for the licensing of physicians, to examine applicants, to set standards for the loss of license, and to withdraw licenses if warranted. They were given the power to tax (but not more than $1.00 from each physician) to pay the costs of licensing activities. Those not licensed could not avail themselves of any legal process for collection of fees, but penalties for the unlicensed practice of medicine were not to come for over 100 years. Standards set by the Society were rigorous in view of the training and education available to physicians at the time.

In retrospect patients were frequently better off in the hands of "quacks," who probably did little harm, than in the hands of educated and trained physicians, using accepted techniques (bleeding, purging, blistering, etc.) often doing more harm than good. Most medical practices of the early 19th century and before were based on unproven theories as to the causes of disease and unquestioned acceptance of treatment methods used by their peers.

We see these same shortcomings in various forms in modern times, sometimes with harmful results. Less than two decades ago physicians often treated hypotensive patients with damaged myocardium and congestive heart failure with hypertensive agents (norepinephrine, at first!). This was the commonly accepted method of treatment, based on the unsound theory that hypotension was more harmful than a failing heart. Diuretics were not used if the blood pressure was below an assigned level; nitroglycerin was avoided for the same reason. No one knows how many deaths resulted from this mistreatment, but certainly it was harmful to many patients. Fortunately, the real problem is now universally recognized, and treatment is aimed at reducing the demand on the weakened heart. Blood vessel dilators (including nitroglycerin) and diuretics are now basic in treatment of this disorder, with minimal concern for asymptomatic hypotension.

Having been given so much control over the quality of physician training and practice, the first CSMS by-laws (adopted in 1793, and two pages long) contained only a brief paragraph on quality of care, as follows:

> Be it ordained, That it shall be highly disreputable for any member to assume or hold the knowledge of any nostrum, or palm any medicine or composition on the people as a secret, and that any such member shall be deemed unworthy to belong to the Medical Society; and that the members of the society shall hold no medical correspondence with such characters, nor consult with them in any medical case whatever; and that all pretenders to nostrums shall be deemed and considered proper subjects for expulsion from this society.

This was taken seriously. Both the county associations and the state Society did expel members for just such reasons. The available records do not name those expelled, or for that matter all those licensed during early years, although a few cases are mentioned. One member had his license returned in six months after his commitment to publish the formula for his magic cure in every newspaper in the state, sending a letter to every physician in the state naming the composition of the nostrum, including an explanation for the use of each substance, and replacing labels with a full description of the composition and results expected from the substances used! Apparently, conning a patient was more serious than medical misadventures.

A major factor in exempting Connecticut from DeVille's deluge in malpractice claims in the middle of the nineteenth century was the immense accomplishments in the advancement of medical care that resulted from giving the Society and its few members so much control over medical care in Connecticut. From the time of their inception, the county and state societies assigned one or more members to give dissertations on appropriate topics at every meeting. Physicians were asked, or almost required, to report cases of scientific interest, particularly if autopsied (usually done by the attending physicians and sometimes in the home). Licensure requirements were increased almost yearly. The Society participated in founding (and for 74 years in operating) the Institute of Medicine at Yale, as well as the first institute for the care of the insane, and the first hospital (in New Haven). Local health departments, with recording of vital statistics, were founded. The Society lobbied the state for a health department (established in 1878 after *50 years* of pressure!). All of this was accomplished before the repeal of "Section 8" of the Society's charter in 1834, effectively removing all medical licensure in Connecticut for the next 57 years.

In 1891 the efforts of the Society finally prevailed in the legislature and a state licensing board was created. The state Society continued throughout this period, in cooperation with the Institute of Medicine at Yale, in giving examinations and granting certificates indicating that the applicant was well prepared and qualified to practice medicine. Physicians from other states, during this 99-year period, applied to the Society and were examined, and, if found qualified, were given a certificate.

The period of quiet in the Society's recorded interest in malpractice ended abruptly in their centennial year (1892) when several papers were given on malpractice in the use of chloroform for anesthesia. An unfavorable and much publicized suit had occurred in Australia, with reverberations throughout the medical world. These several reports indicated that the threat of malpractice was having a dramatic effect on medical practice in Connecticut, even though chloroform had been used extensively and with confidence.

Cited in one of these papers by Dr. William Wainwright[11] was a statement at the International Congress at Berlin in 1890 from a Dr. Wood. Dr. Wood affirmed that "...deaths from chloroform are preventable; that with due care they may be avoided; and that therefore when they occur they are the result of ignorance or carelessness. Five hundred surgeons, including such names as Billroth, Jaeger, McLeod, Simpson, Agnew, McGuire, Hunter, and others of equal rank [are] guilty of manslaughter *and still the carnage goes on*."* On the other hand, Dr. Wainwright quoted some American statistics "...Dr. Henton McGuire reported 28,000 chloroform administrations in Stonewall Jackson's army without a single fatality ....Dr. Nussbaum reported 40,000 administrations without a death. ...[Dr.] Chisolm reported 15,000 without a death." None of these articles in 1892 demonstrated awareness of the nature or existence of toxic reactions to chloroform. The army surgeons' statistics probably have little authenticity in view of this lack of knowledge. Of the greater importance are the comments in the House of Delegates by Dr. Stern, discussing Dr. Wainwright's article, "In all the relations of life the questions of responsibility are coming more and more to the front. Individuals, towns, cities and common carriers are holden responsible for whatever negligence contributes to the harm of others. No profession stands so much in jeopardy as our own ...the public even demands that the court shall decide that a surgeon must warrant the result of an operation!"

---

*Discussed in such a forum and in such dramatic prose it is no wonder the subject reverberated throughout the medical world and even into Connecticut!

The increasing awareness of the threat of malpractice associated with the use of chloroform (previously used extensively with presumed safety) not only increased physician perception of the rising incidence of malpractice claims but also emphasized the rapid spread of the malpractice threat from the treatment of fracture almost exclusively to every field of medical care. DeVille reports that 90% of 19th-century malpractice claims involved fracture care. The remaining 10% consisted mostly of complications of small pox vaccinations and amputations. He also reports that prior to 1820 the usual treatment, supported by most authorities, of compound fractures of the extremities was amputation. Attempts to preserve the extremities led to less than perfect results and to the first proliferation in malpractice claims of the 19th-century. Literature and records of that time indicate that some physicians refused to treat fractures, and others reverted to amputations, in order to avoid malpractice suits. This early history confirms several principles not usually considered characteristic of the effects of the threat of malpractice on medical practice. The first was that improvements in quality (ie, saving an extremity) led to increased threat of malpractice, and secondly this led to a retreat to accepted poorer treatment, and thirdly that most claims were in an area (bone-setting) in which physicians were most proficient. A fourth principle might well be that the increasing quality of medical education and the explosion in the science of medicine that were taking place at the turn of the century resulted in corresponding increases in malpractice litigation. Thus, while celebrating its centennial, the society was being introduced into the new world, not only of expanding scientific knowledge, but also of increasing legal responsibility.

Twelve years later, in the *Journal of the American Medical Association (JAMA)*, 4 January 1902, an unnamed New York State representative reported that on 14 December 1901, the New York County Medical Association added a branch for the defense of its members against malpractice suits and approved a plan wherein the executive committee, when requested, and upon compliance with certain provisions, would assume the defense of suits for alleged malpractice brought against its members. The committee then took full charge of the defense, furnishing all legal services, expert witnesses, and all necessary expenses. They would not compromise except with the physician's consent. However, the society was not obligated for the payment of any damages awarded by the courts or agreed to by compromise.

This *Journal* included an editorial favorable to organized medical defense. The editor commented, "What affects the honor and standing of one member affects the honor and standing of the entire profession," and that "there is no question but that an organized effort on the part of the profession will have a tendency to deter blackmailers from bringing suit and discourage pettifogging lawyers, ..." The editor also spoke out strongly against compromising (settling) a case. Also in 1902 the Chicago Society started its own legal department for the same purpose, and in 1905 the Wayne County Medical Association approved creation of a "Defense League" for Detroit physicians. The editors of *JAMA* in their brief comments do not indicate whether any of the early defense activities or societies insured against indemnity losses. As noted later, there were probably no commercial insurance companies writing such liability policies until at least 1911.

The Connecticut State Medical Society's first significant response to the malpractice issues raised in the centennial did not come until 22 years later. In May 1915 Dr. William R. Miller of Southington gave a report, in his retirement speech as president of the Hartford County Medical Association, on medical defense in other states. Motivated by a two-fold increase in malpractice claims in Connecticut in the previous two years, "...probably as a result of the new Workmen's Compensation laws that forced ambulance-chasing lawyers to look for new fields not exempt from their efforts at extortion," Dr. Miller obtained data from 24 other state medical societies that were protecting their members against suits for malpractice. The following is excerpted from Dr. Miller's later comments to the CSMS house of delegates:

> We naturally expect that suits for malpractice are expensive. ... In all the states of the Union the cost per member has been less than one dollar per year....The average in all states, when figures were given, was $120 per case....From the 21 states reporting this item, 779 cases have been tried and but 10 lost. Approximately but one adverse decision in 80 cases passing through the court. Of course this is the number of cases which actually came to suit. The number of cases which did not come to the defense committee is not known. Many cases were settled by physicians themselves. It is estimated the state societies themselves settle, or at least throw out of court or prevent coming to trial, 80% of all cases that come before them. This does not mean that they pay any damages. They simply, by protecting the physician, put up such a stiff fight that the case is dropped. That is one of the strong features of medical defense by state societies that no cases are settled, every case is fought and appealed if necessary.... Because of this the lawyers are much more careful about bringing suit....
>
> The New York State Medical Society has defended 147 cases in the last 12 years without the loss of a case. Iowa has defended 54 in the last seven years without a loss; and Michigan 125 during the last six years with but one adverse verdict.[12]

Records of cases not defended by the several societies are unavailable. Of the several defense committees on which more complete information is available, all defended only those cases that were judged by the committee as deserving of defense. The selection of cases and the considerable influence over expert witnesses help to explain the phenomenal success of the defense committees in the early part of the 20th-century. Even though there may have been a fair number of suits that deserved only a proper settlement, the defense activities of these societies must have discouraged frivolous suits and educated physicians, hospitals, and societies in risk management. Dr. Sandor's records of appellate cases[13] do not show a significant decrease in rate of claims during this period. Rather, the decade from 1920 to 1930 had by far the highest incidence for the country as a whole with 470 suits(208 losses) compared to 285 suits (137 losses) in the 1945 to 1955 decade, and only 224 suits with 103 losses in the entire time prior to 1900.

Dr. Miller's motion in the House of Delegates to create a study committee on medical defense was approved. The president appointed Dr. Miller (chairman), Dr. Everett J. McKnight, and Dr. Frank H. Wheeler to the committee. In its report to the CSMS annual meeting in 1916 this committee recommended change of the by-laws which would create a Committee on Medical Defense with three members and the secretary of the Society. The committee would investigate all claims against members and take full charge of all cases considered proper for defense, including providing defense lawyers, expert testimony, and all costs of defense, but not obligating the society to any indemnity losses. The annual dues would increase by one dollar, this sum to be set aside in a medical defense fund.

Because of Dr. Miller's absence and the concerns of the First World War neither the Committee nor the Society took action on the proposals until 1920, when Dr. William H. Donaldson of Fairfield was appointed chairman. At the following meeting the recommendations made by Dr. Miller's committee five years previously were reintroduced. With slight modifications, (the provision for an increase in dues was deleted) the proposal was approved. Later in 1920 Dr. Miller, who had returned from military service and resumed the chairmanship, and the committee prepared, and the council approved, an action plan. Reports were given at all county meetings, all members were to be circularized, and Messrs. Day and Berry of Hartford were retained as the committee's attorneys.

At the Society's next meeting Dr. Miller reported that no appeal for defense had yet been made, though the Committee had been of service in the prevention of some suits and also of some aid in already pending suits.

Of interest in terms of present day risk management programs is that the circular sent to society members 70 years ago mentions, "...the bad effect of indiscrete or disparaging remarks...difficulties caused by failure of doctors to keep proper records...where the patient refuses to abide by the instructions...such instructions be written and witnessed...that x-rays should always be taken before and after treatment of fractures...the need for written consent before procedures...signed consultant statements...the need for definitely knowing that all packing material and swabs have been removed...," (counting of instruments and materials had not yet been initiated and Dr. Miller proposed the attachment of some radiopaque material to all radiolucent materials) and, "...that all physicians keep abreast of the times." Also in this circular is the first mention of professional liability insurance. The Committee reported that by then (1922) each county society had available to it group liability insurance with Aetna Life Insurance Company.*

> Every possible means has been provided for the most effective co-operation between the committee counsel and legal department of the Aetna Life Insurance Company in the actual investigation of claims and preparation and trial of cases, thereby making available the excellent machinery provided by the insurance company for this purpose. No settlement of claims will be made without the consent of the doctor affected and suitable safeguards are provided against improper settlements. This whole plan has been investigated carefully by the Council of the Medical Society of the State and is recommended as satisfactory by them.

Insurance was necessary since, if the defense was unsuccessful, the Society was not liable for indemnity payments, and also the Society did not defend justifiable suits. A large proportion of members did not subscribe to insurance until some years later. The only figures available record that 598 of almost 1190 members were insured by Aetna in 1922.

The new by-laws required reporting to the society all claims and suits, but no statistics on reported, defended, settled, wins or, losses have been found. Cost figures include only the costs to the Society and \ or defendants of claims and suits defended by the Committee. In 1922, the first year of full operation, the Committee reported six cases either terminated or in preparation with a total of $150 for a legal fee and $17.50 for stamped envelopes.

Late in 1923 Dr. Miller died and was replaced by Dr. Frank H. Wheeler of New Haven. Dr. Wheeler's report of May 1924, notes a number of claims and suits, of which 13 had been terminated, one for a small amount and the other for $750 (about $20,000 in 1992 dollars). In the 1925 report, made by Dr. Ernest A. Wells of Hartford, now chairman, the Committee had been involved in 28 cases during the year. Total losses, all for indemnity payments on three cases, came to $1,905. Dr. Wells also commented that committee members made no charges for their time or postage, stenography, stationery, etc., which were minimal, and therefore there was no expense for the year's work.

One case was of great interest and was therefore reported. Occurring in 1918, almost six years prior to the suit, it involved a sponge left in an arm. The jury awarded $2,000 but the case was appealed to the Connecticut Supreme Court. The outcome was not salutary. The Court

---

*As part of its centennial celebration Aetna Life Insurance Company printed a booklet entitled *Aetna Life—The First 100 Years*. Aetna Life was chartered to write liability insurance in 1893, but it was not until 1902 that new lines of liability insurance, including "employers liability" and "workmen collective insurance," were developed. In 1907 a dormant company writing mostly auto insurance liability was acquired. In 1911 Aetna added, through this company, "public liability," "general liability," and "elevator liability." Found recently by archivist Paul Lasewicz at Aetna is a publication sent by Aetna Life to agencies on 15 March 1915 which describes two professional liability policies available from Aetna Life. One policy covered the defence of malpractice suits, the other covered indemnity losses with limits of either $10,000-25,000 or $5,000-15,000. Premiums were not mentioned. Searches of similar literature from the Phoenix Mutual Insurance Company and the Travelers Insurance Company were even less revealing, although they, as well as Liberty Mutual, are mentioned in later CSMS records. No further information about premiums, coverage, or policies during or prior to the first two decades of this century have as yet become available to the author.

ruled that the contract aspect of a suit against a physician is not limited by the one-year statute of limitations, thus making the distinction between tort and contract law more vague. The six-year limitation in contract law became more threatening.

Notable was a comment in 1925 in the president's remarks, which also supported a professional journal and a full-time executive secretary, that, "The general service of our society to its members is not all it should be, except in the matter of medical defense."*

In 1928 Dr. Donald B. Cragin of Hartford succeeded as chairman of the Committee. Dr. Cragin reported the cost of closed cases from 1 January 1927 to May 1928 to be $11,256.87. In this same period 19 cases were considered closed, and 16 cases were still outstanding (five originated before 1927).

An issue which had not been previously mentioned in the "Proceedings" appeared in the form of a letter from J. A. Howard, a member of the legal firm of Day, Berry, and Reynolds, attorneys for the defense committee, at the 1928 annual meeting. Dr. Howard reported:

> When Dr. W. R. Miller (first chairman of the Committee on Medical Defense) first spoke to me about undertaking malpractice defense for the state Society, he gave me to understand that members of the Society had all agreed to donate their services as experts when called in defense of any fellow member. This was repeated in several conversations with me and was the understanding that Dr. Weston had at the time.
>
> I wonder whether you would be willing to look up this question in your records because several doctors have presented bills in malpractice cases and, if the question has not already been settled, it seems to me that it should be.
>
> From my standpoint I should much prefer that doctors make no charge for expert testimony in defense of malpractice cases because ... a witness may be asked, in cross-examination, whether he is being paid by the defendant or his attorney. If he can answer that he is not paid and expects no pay, he is worth five or six paid witnesses....[14]

Mr. Howard continued with a number of other arguments supporting his view and pointing out that "It would be unusual for even the most widely known and respected expert in any field to have to appear more than once in his lifetime. Such experts had the most to gain since they and their referring physicians were the most likely targets of suits." The House of Delegates then adopted the following:

> Resolved, that the House of Delegates of the Connecticut State Medical Society, during the 136th Annual Meeting, in Bridgeport, 23-24 May 1928 states and affirms as a policy that each member of said society shall give medical testimony, including 'expert testimony,' in the mal-practice-suit case of any defendant fellow-member, without fee, reward, or expectation thereof; such refusal of remuneration being solely for the better protection of and the mutual helpfulness within the profession.†

This decision was obviously intended to apply only to cases against physicians. At this same meeting, the House received a report that:

> The Court of Common Pleas has ruled, through precedent of a case decided in 1857, that since a commissioner of the Superior Court, by subpoena, has the power on payment of ordinary witness fees, to bring in any witness and compel him to state anything that is in his knowledge, and agreement to pay fees in excess of the legal fees (60¢) is without consideration and cannot be recovered in suit...."

---

*The commendation would have been worth more if the rest of the Society had recognized the value of the work being done by this committee.

†Found in Middlesex County records, probably for 1914, was a note from the state secretary asking that the county act on resolutions concerning the "Leeds' methods" of conducting expert testimony. These resolutions called for "... preliminary consultation by medical witness on both sides of the case as to its status ... contingency fees by physicians were not in accordance with medical ethics...." No reference to these resolutions was found in the proceedings of the state Society.

The House of Delegates then directed the Committee on Legislation, already busily lobbying for changes in the statute of limitations on contract cases, to "Endeavor to secure enactment by the 1929 General Assembly new laws or changes in existing laws whereby any agreement made by a party to a suit, or his representative, with an expert (medical) witness, should be enforceable by law." The changes sought were passed by the legislature and the Society approved payment of a (lobbyist's) fee to Day, Berry, and Reynolds for their help.* No evidence of rescinding the policy on expert testimony has been found. However, as the Society soon withdrew from involvement with medical defense, the practice of appearing as an expert witness, without charge, in defense of an accused physician has long since been forgotten. Instead, some physicians expect to profit greatly at the expense of fellow physicians.

Dr. Cragin had given very complete reports in 1928 and 1929, including summaries of all cases of alleged negligence which were under consideration, with outstanding expenses and damages.[15]

The summaries were as follows:

	1928	*1929*
Claims outstanding—	4	2
Suits outstanding—	15	14
Claims arising—	15	16
Suits arising—	n/a	10
Defense expenses paid—	$ 6,992.39	$ 4,162.74
Damages paid—	$ 9,912.13	$ 2,700.00
Outstanding estimates as of December—	$13,756.00	$17,496.00

In spite of the success of the Society's Committee on Medical Defense, both in management of cases and in risk management, the following surprising report was given in the May 1930 annual meeting by the councilor from New Haven County:

> An unprecedented number of malpractice suits against members of our Association have been instituted recently so that high insurance rates would have to be charged for protection unless something was done to discourage such suits. To this end, the New Haven Medical Society appointed a Committee, which, after careful study of the situation, recommended that a permanent Committee be appointed, the duties of which include a careful examination into the merits of any threatened suit, so that the rights of both the patient and the physician shall be safeguarded. Then, when all available facts are in its possession, the Committee shall recommend either settlement of the suit or vigorous opposition to it. The decision of the Committee is binding upon our members with unhappy results promised to any who may defy it.
>
> Following the action of the New Haven Medical Society the New Haven County Medical Association has voted similar measures, and it is believed that an important movement has thus been launched.

No statistics were quoted and the available information does not support these contentions; instead, statistics confirm a rise ensued immediately after the institution of workmen's compensation laws 15 years earlier, but there was no significant rise in the incidence of malpractice during the decade before 1930.

---

*In 1969 the statute of limitations was extended to two years beyond discovery, but in no case more than three years, for all malpractice suits.

†Again no supporting statistics were given.

‡This did not occur. There were prctically no increases in the next 15 years, nor were there reasons to expect them during the depression years.

The New Haven County plan duplicated the successful 15-year-old CSMS Committee on Medical Defense but on the county level. The new county level plan differed only in *requiring* every member to comply with the county society's directives.

Even more surprising was a statement to the House from the Council, formulated at a special meeting on 21 May 1930. In this remarkable report the Council states that malpractice had "materially increased."† They predicted a four- to six-fold increase in insurance premiums.‡ They recommended all aid by the Society staff and physicians for defendant physicians be discontinued. Physicians were to rely solely on insurance. The Council speculated about the Society's rising budget at the same time stating, "Dues have never been increased because of medical defense." They felt that the state Society should not "require" insurance of members in order to obtain society aid for their defense, but then supported the New Haven County plan which did just that!

They recommended *another* committee be appointed to study the subject and at the same time recommended by-law changes to abolish the Committee on Medical Defense.

They suggested that this would save money for members, at the same time approving the New Haven plan which transferred expenses from the state to the county level! The House of Delegates approved the Council's recommendations. In their meeting of 23 January 1931, the Council received a report (not available in the proceedings) of the special committee on medical defense planning, and recommended its adoption by the various county associations.

The Council also decided not to nominate members to the soon-to-be-deleted committee on medical defense. They proposed the creation of a special committee on "Medical Ethics and Deportment," recommending that each county do the same. Such committees would review and make recommendations on malpractice cases referred to them.

The report given to their annual meeting by the committee from New Haven mentioned that important measures being considered by the interim committee would be put into effect at the fall meeting. The report from the soon-to-be-defunct Committee on medical defense, given by Dr. Cragin to the May 1931 annual meeting of CSMS, included 15 new claims and six new suits in 1930, with total paid damages and expenses for the year of $6,485.90 and estimates of future losses on outstanding claims and suits of $4,150.* CSMS dues remained at $4.00, and the by-law changes eliminating the Committee on Medical Defense were approved. This effectively ended CSMS involvement in the area of malpractice.

In 1932 a committee was appointed to study the subject of "blanket" insurance. The by-laws were amended to provide for the Committee on Ethics and Deportment consisting of the chairman of each county committee and the president and secretary of CSMS. The committee was to "Investigate and advise" in malpractice cases referred to it by county committees on ethics and deportment, but it was to take no part in legal defense. Discussion included the optimistic opinion that "If reputable lawyers are convinced that honest opinions result from reference to these committees they will cooperate even to the extent of disciplining unscrupulous members of their own profession who attempt to bring nuisance suits." In 1933 the new Ethics and Deportment Committee offered a many-paged report as to how their committee would operate, with much discussion as to whether their workings should be available to the courts. Some discussants thought the courts might benefit from having "the attitudes of the medical profession regarding malpractice suits."

The idea that group malpractice policies might solve the whole problem was accepted, and, in 1933, the president appointed a "Committee on Group Malpractice Insurance," chaired by Dr. Creighton Barker. Over the next two years Dr. Barker reported that standardization of rates for malpractice insurance throughout the state and the creation of county society group policies had been accomplished with the Aetna Insurance Co. In the mean-time the Committee on Ethics and Deportment reportedly had two cases referred for review but no

---

*Note the *decrease* from the previous two years!

report of this is in the proceedings, and in 1934 the Committee reported, "Nothing referred; no action." CSMS had effectively withdrawn from any responsibility in the field of malpractice except to obtain insurance for members.

In the 1944 annual meeting the problems of group insurance were again brought up, this time by New London County Medical Association, where rates seemed to vary inexplicably and group insurance was available to two smaller societies, but not to all county association members. Again, a committee was formed and again found wide variations in policy rates and coverage throughout the state, even with Aetna. No coverage was provided for cases tried under contract law but the basic $12.50 premium was, as Dr. Barker stated, the "lowest in this part of the country." He also referred to a state statute adopted a few years before 1944 "Establishing a limit of $15,000 in damages involving a death case." His committee negotiated a statewide plan with Aetna with premiums of $12.50 for $10,000 / 15,000 coverage with increased premiums for limits as high as $35,000. Cases tried under contract law would also be covered at no extra cost.

By 1970 premiums were skyrocketing, and it was obvious that abandonment of the Society's role in the management of malpractice did nothing to control costs. Reasonable physicians should not have expected that entrusting the management of malpractice to insurers, whose economic interests are diametrically opposed to those of the physicians, could result in either lower expenses or lower incidence of cases.

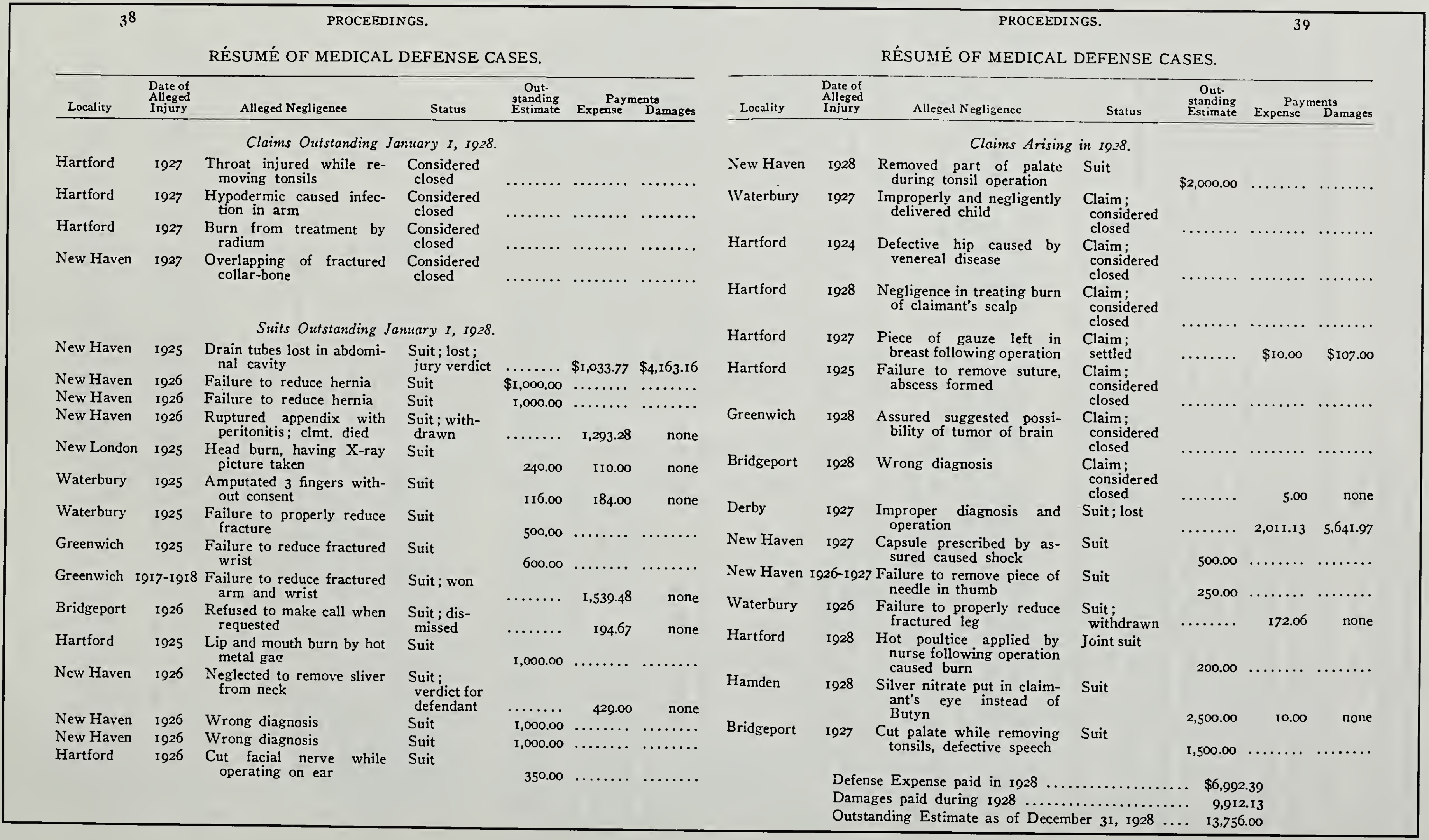
It would be of considerable interest to know the fate of the medical defense committees in the other 24 states mentioned by Dr. Miller in 1915, and in the two county associations (Chicago and New York) with histories beginning in 1902. Though there seems to be little in the available literature, here and at the AMA, an interesting but time-consuming project would be to visit many of the state organizations where society records would probably be informative.

It is interesting to note that the knowledge and foresight leading to involvement of CSMS in the management of malpractice, originating with Dr. William R. Miller, was lost soon after his death. Although in Connecticut this brief excursion into a mostly ignored professional responsibility was almost entirely due to this one extraordinary physician, there must have been *something in the air* which would cause 24 other societies to follow the same path in the same period. One wonders if Dr. Miller and his confreres envisioned that 60 years after his death over 60% of American physicians would be participating in society-founded organizations assuming all the responsibility for handling this affliction of our profession.

REFERENCES

1. DeVille KA: *Medical Malpractice in 19th-Century America*, New York University Press, 1990.
2. Markby: *Elements of Law*, 6th edition, Oxford University at Clarendon Press, 1906.
3. Sandor AA: *The history of professional liability suits in the United States*, JAMA 1957; 163:459.
4. Britton: *Homicide*, circa 1290, edited and translated by F.M. Nichols, London, Macmillian cs, 1865, 6 p. 34.
5. Fitzherbert A: *Natura Brevium*, London, Richard Totell, 1553, cited by (3) p. 459.
6. Y.B., 48th Ed. III.; f. 6, pl. 11, cited by Sandor (3) on p. 459.
7. *Oxford English Dictionary*; C.T. Onions, Oxford University at Clarendon Press, 1955.
8. Op. cit. #3., pp 461-5.
9. Cross *vs* Guthrie, 2 Root 90, Connecticut, 1794.
10. Op. cit. #1, pp 2-5, 35-41.
11. Wainwright W: The medico legal aspects of chloroform, *Proceedings of* CSMS, May 1892, pgs. 377-83.
12. Miller W: Comments, *Proceedings of* CSMS, May 1915, pp. 77-9; and Dr. William Miller *Medical Defense against suit for malpractice*, delivered to Hartford County Medical Association, probably in 1914 but undated.
13. Op. cit. #3, pp 461-3.
14. Howard JA: Remarks, Proceedings of CSMS, May 1928, pp 79-80.
15. The summary is intriguing and informative; it is reproduced from the *Proceedings of the Society* of May 1929, pp 38-9.

Summaries of All Case of Alleged Negligence, 1928-29[15]



38 PROCEEDINGS.

RÉSUMÉ OF MEDICAL DEFENSE CASES.

Locality	Date of Alleged Injury	Alleged Negligenee	Status	Out-standing Estimate	Payments Expense	Payments Damages
		Claims Outstanding January 1, 1928.				
Hartford	1927	Throat injured while removing tonsils	Considered closed	....	....	....
Hartford	1927	Hypodermic caused infection in arm	Considered closed	....	....	....
Hartford	1927	Burn from treatment by radium	Considered closed	....	....	....
New Haven	1927	Overlapping of fractured collar-bone	Considered closed	....	....	....
		Suits Outstanding January 1, 1928.				
New Haven	1925	Drain tubes lost in abdominal cavity	Suit; lost; jury verdict	....	$1,033.77	$4,163.16
New Haven	1926	Failure to reduce hernia	Suit	$1,000.00	....	....
New Haven	1926	Failure to reduce hernia	Suit	1,000.00	....	....
New Haven	1926	Ruptured appendix with peritonitis; clmt. died	Suit; withdrawn	....	1,293.28	none
New London	1925	Head burn, having X-ray picture taken	Suit	240.00	110.00	none
Waterbury	1925	Amputated 3 fingers without consent	Suit	116.00	184.00	none
Waterbury	1925	Failure to properly reduce fracture	Suit	500.00	....	....
Greenwich	1925	Failure to reduce fractured wrist	Suit	600.00	....	....
Greenwich	1917-1918	Failure to reduce fractured arm and wrist	Suit; won	....	1,539.48	none
Bridgeport	1926	Refused to make call when requested	Suit; dismissed	....	194.67	none
Hartford	1925	Lip and mouth burn by hot metal gag	Suit	1,000.00	....	....
New Haven	1926	Neglected to remove sliver from neck	Suit; verdict for defendant	....	429.00	none
New Haven	1926	Wrong diagnosis	Suit	1,000.00	....	....
New Haven	1926	Wrong diagnosis	Suit	1,000.00	....	....
Hartford	1926	Cut facial nerve while operating on ear	Suit	350.00	....	....

PROCEEDINGS. 39

RÉSUMÉ OF MEDICAL DEFENSE CASES.

Locality	Date of Alleged Injury	Alleged Negligence	Status	Out-standing Estimate	Payments Expense	Payments Damages
		Claims Arising in 1928.				
New Haven	1928	Removed part of palate during tonsil operation	Suit	$2,000.00	....	....
Waterbury	1927	Improperly and negligently delivered child	Claim; considered closed	....	....	....
Hartford	1924	Defective hip caused by venereal disease	Claim; considered closed	....	....	....
Hartford	1928	Negligence in treating burn of claimant's scalp	Claim; considered closed	....	....	....
Hartford	1927	Piece of gauze left in breast following operation	Claim; settled	....	$10.00	$107.00
Hartford	1925	Failure to remove suture, abscess formed	Claim; considered closed	....	....	....
Greenwich	1928	Assured suggested possibility of tumor of brain	Claim; considered closed	....	....	....
Bridgeport	1928	Wrong diagnosis	Claim; considered closed	....	5.00	none
Derby	1927	Improper diagnosis and operation	Suit; lost	....	2,011.13	5,641.97
New Haven	1927	Capsule prescribed by assured caused shock	Suit	500.00	....	....
New Haven	1926-1927	Failure to remove piece of needle in thumb	Suit	250.00	....	....
Waterbury	1926	Failure to properly reduce fractured leg	Suit; withdrawn	....	172.06	none
Hartford	1928	Hot poultice applied by nurse following operation caused burn	Joint suit	200.00	....	....
Hamden	1928	Silver nitrate put in claimant's eye instead of Butyn	Suit	2,500.00	10.00	none
Bridgeport	1927	Cut palate while removing tonsils, defective speech	Suit	1,500.00	....	....

Defense Expense paid in 1928	$6,992.39
Damages paid during 1928	9,912.13
Outstanding Estimate as of December 31, 1928	13,756.00

For certain categories of taxpayers or people on extension, deductions for 1992 might still be available.

ENDORSED BY THE MEDICAL SOCIETY OF NEW JERSEY

**V.E.B.A. PLAN*

FEATURES

TAX
DEDUCTIBLE
CONTRIBUTIONS
TO AN
I.R.S. APPROVED
TRUST

EVEN IF ...

- **YOUR PENSION PLAN IS OVER FUNDED**
- **YOUR PENSION PLAN IS MAXIMUM FUNDED**
- **YOU HAVE NO PENSION PLAN**

PLEASE SEND ME A copy of your informational pamphlet on the * V.E.B.A. program.
I understand there is no obligation.

NAME______________________________

ADDRESS______________________________

DATE OF BIRTH________________ TELEPHONE # (____)________________

Mail to:

THE KIRWAN COMPANIES
402 MIDDLETOWN BLVD., SUITE 202
LANGHORNE, PA. 19047

(800) 283-7666......................... (215) 750-7616......................... FAX (215) 750-7791

*VOLUNTARY EMPLOYEE BENEFICIARIES ASSOCIATION

The Crisis in Correctional Health Care: The Impact of the National Drug Control Strategy on Correctional Health Services

AMERICAN COLLEGE OF PHYSICIANS,
NATIONAL COMMISSION ON CORRECTIONAL HEALTH CARE,
AND AMERICAN CORRECTIONAL HEALTH SERVICES ASSOCIATION*

> *We therefore conclude that deliberate indifference to the serious medical needs of prisoners constitutes the "unnecessary and wanton infliction of pain" ... proscribed by the Eighth Amendment.*
>
> U.S. Supreme Court, 1976[1]

THE United States now incarcerates a greater percentage of its population than any other country: for every 100,000 persons, 426 are confined in prisons and jails.[2] Most inmates are male, young, poor, and from minority groups. Most are substance abusers with substantial physical and mental health needs.

In the past decade, prison and jail populations doubled,[3,4] outpacing all attempts to increase the correctional system's ability to house and care for these inmates. From 1979 to 1988, direct governmental expenditures on corrections increased 216%.[5] Despite this increase, at the end of 1990, prisons nationwide were estimated to be 18% to 29% over their capacities, while the federal system operated at 51% over capacity.[3] Jails were operating at 104% of their rated capacity.[4] The entire prison systems of 10 states, major institutions in another 30 states,[5] and 30% of all large jail jurisdictions[4] are under court order to improve conditions.

These problems in our criminal justice system have been exacerbated by federal initiatives to reduce the use of illegal drugs. The most recent initiatives, known as the National Drug Control Strategy (NDCS), were first announced in September 1989 and have been updated yearly. The NDCS emphasizes mandatory minimums for many drug crimes. In 1989, the National Council on Crime and Delinquency predicted that "the current War on Drugs will overwhelm the nation's correctional systems over the next five years."[6] The 1991 NDCS notes, "As the recommendations of the criminal justice portion of the Strategy are implemented, prison overcrowding is likely to continue, posing difficult policy choices."[7]

Members of our organizations are devoted to health care and see firsthand the health effects of these difficult policy choices. Corrections in general and correctional health care in particular have suffered negative consequences: severe overcrowding, insufficient programs to treat substance abuse, and increasing prevalence of communicable diseases such as the acquired immunodeficiency syndrome (AIDS), tuberculosis, and hepatitis. The large increase in the number of substance abusers and sick and terminally ill inmates has rendered our nation's prisons and jails physically or financially unable to deal with their current populations, much less the explosive increases the future holds. It is the magnitude of inmate health problems that threatens to overwhelm the substantial gains made in correctional health care over the past two decades.

As measured by recognized standards of inmate health and health services, our correctional systems are in crisis.

Reprinted from the *Annals of Internal Medicine* 1992; 117:71-7.

*This paper, authored by Janet Weiner, M.P.H. and B. Jaye Anno, Ph.D. was developed by the American College or Physicians' Human Rights and Medical Practice Subcommittee for the Health and Public Policy Committee. Members of the Human Rights Subcommittee for the 1991-92 year are: Quentin D. Young, M.D., Chair; Carola Eisenberg, M.D.; William G. B. Graham, M.D.; Herbert Rothenberg, M.D.; Kim Thorburn, M.D.; and Abraham Verghese, M.D. The American Correctional Health Services Association's Board of Directors approved it in February 1992, the American College of Physicians' Board of Regents approved it on 24 March 1992, and the National Commission on Correctional Health Care's Board of Directors approved it on 30 March 1992.

Acknowledgments: The authors thank the three organizations that developed this paper for their extraordinary cooperation, especially Kim Thorburn, M.D., president of the American Correctional Health Services Association; Robert Burmeister, Ph.D., president of the National Commission on Correctional Health Care; and Ellen Marshall, policy coordinator of the American College of physicians.

Requests for Reprints: Linda Johnson White, Director, Department of Scientific Policy, American College of Physicians, Independence Mall West, Sixth Street at Race, Philadelphia, PA 19106-1572.

As a nation, we must respond to the problem of drug abuse with rational strategies that do not overwhelm the capacity of our criminal justice system to care for its inmates. (For a definition of terms used in this article, see Appendix.)

Background

The State of Correctional Health Care.—Recent improvements in correctional health services are threatened by the nation's burgeoning prison and jail populations. Beginning in the 1970s, civil rights litigation and involvement by organized medicine resulted in the establishment of community standards in prison and jail health services where neglect had been the norm. Another positive step was the implementation of accreditation standards for correctional facilities by the National Commission on Correctional Health Care (NCCHC). The standards promote adequate health care systems through voluntary accreditation. To gain NCCHC accreditation, correctional facilities must meet standards for intake, assessment, and screening of incarcerated persons. In 1991, the NCCHC accredited 73 prisons (11%) and 230 jails (7%).

These standards have become increasingly difficult to meet and maintain during this period of population growth. Health services are a costly component of correctional facilities' operating budgets, taking up from 2.8% to 18.9% of state correctional department budgets.[8] As more persons with greater health needs are incarcerated, this percentage is likely to increase. However, correctional health care budgets apparently are not increasing in proportion to the growing numbers of inmates with significant health problems.[9]

No staffing formulas for health services in correctional institutions have been reported in the literature. The NCCHC standards specify one full-time equivalent physician for every 750 to 1,000 inmates, but the Commission recognizes that no simple formula exists for determining appropriate staff size. The number and type of health care personnel required by an institution depends not only on its average daily population but also on the total number of inmates received during the year, their lengths of stay, and their particular health care needs.[10] This lack of precise staffing formulas limits the ability of institutions to ensure appropriate expenditures for health care personnel as facilities are built or expanded. As this problem becomes more acute, expenditures for health services and medical supplies also may be jeopardized. The current situation encourages an unhealthy competition among security, housing-sustenance, and health care requirements for the same limited resources.

In addition to being ill-equipped for the population explosion, most correctional health services are not set up to meet the extensive needs of inmates. The typical delivery model is a reactive system of care on demand: sick call. Health services are requested rather than scheduled. Such a system does not lend itself to regular follow-up care, which is needed to manage chronic disease. Primary care and prevention models of health care delivery have not been emphasized in correctional systems. Even when health screening is done, systems may not be capable of following up on abnormal findings.

An ongoing problem in correctional health care is the lack of comprehensive data to define more accurately the population served and its health care needs. This problem has both public policy and medical professional components. Policymakers have not committed the resources to foster research in this setting or to develop academic programs that would train medical professionals in correctional health care. Medical professionals, for the most part, have not stressed the placement of correctional health care within the public health sector and have not forged extensive linkages with public health professionals. In general, public health professionals have avoided including correctional health in their sphere of concern.

We are aware of successful collaborative ventures among academic medical centers, public health departments, and correctional institutions. One such effort in 1990 resulted in an effective program for human immunodeficiency virus (HIV) counseling, screening, and treatment in prisoners in Rhode Island (Dixon PS, Flanigan TP, De Buono BA, Laurie JA, Scott HD, Carpenter CCJ: "HIV infection in prisoners: Meeting the health care challenge." Unpublished data). An HIV management team on site includes an attending physician from the Brown University faculty, a rotating infectious disease fellow, a full-time registered nurse, and two AIDS education specialists. This program was made possible with funding from the State Department of Health. Everyone wins in this collaborative arrangement. The prisoners receive ongoing, continuous care; the prison saves expensive, outside visits to specialists; the health department gains a way to reach a high-risk population with preventive education and early treatment of HIV infection; and the academic medical center benefits by providing a unique training site for its fellows.

Such cooperation is essential if correctional health care is to meet the challenges it now faces in providing the community standard of care to all incarcerated people—a challenge made more daunting by the increase in numbers of inmates and the problems they bring with them .

Correctional Populations and Demographics.—On any day in the United States, more than 1.2 million Americans are behind bars. At last count, there were 771,243 men and women in state and federal prisons,[3] 405,320 in jails,[4] and an additional 56,123 in public juvenile facilities.[11] In any year, an estimated 22 million admissions and discharges occur in correctional facilities in the United

States. In 1989, admissions and discharges totalled 20 million in jails[4] and more than 1.2 million in public juvenile facilities;[11] in that same year, prisons admitted 467,227 persons and released 386,228.[12]

In adult institutions in 1990, 94.3% of prison inmates and 91% of jail inmates were male. The proportion of women is growing in both jails and prisons. In the past decade, the number of women in prison tripled, and the overall prison population doubled.[13]

The average age of inmates admitted to prisons in 1989 was 29.6 years.[12] In 1989, 75% of jail inmates were between the ages of 18 and 34 years.[14] However, the number of older inmates is growing. About 5% of those incarcerated in prisons in 1989 were 50 years of age or more.[12]

Ethnic minorities are disproportionately represented in our correctional facilities. Blacks (12% of the U.S population) now constitute 47% of the prison population[5] and 47% of the jail population.[4] For Hispanics (7% of the U.S. population), the corresponding statistics are 9.6%[12] and 14%,[4] respectively.

Growth in Correctional Populations.—The population growth in the nation's correctional facilities has been explosive and is predicted to continue. Much of the growth is attributable to society's concern about drug abuse and attendant crime. Arrests for all offenses rose 27.7% in the last decade, but arrests for drug violations rose 125.9%.[5] Many jurisdictions altered their sentencing guidelines and implemented mandatory sentences, which meant that more convicted persons received incarceration. A concomitant decrease occurred in the use of alternatives to incarceration, such as early release programs. Further, use of fixed sentences increased, which meant that more inmates were staying longer behind bars. At the same time, many jurisdictions deemphasized or abolished early release programs, such as parole.

Because of these policies, by 1989 one in three women and one in five men in local jails were being held on drug charges.[15] Three quarters of them were black or Hispanic. In 1990, 54% of all federal prisoners were incarcerated for drug offenses.[5] Focusing just on state prisons, the National Council on Crime and Delinquency predicted the following:

1. Under existing policies, the states will increase their prison populations by over 68% by 1994, an annual average growth rate of about 13% per year.

2. The projected increase translates into an additional 460,000 prison inmates by 1994 for a total of 1,133,000 inmates. With average operating costs of $25,000 per inmate per year and a construction cost of $50,000 per cell, states will require at least an additional $35 billion to build and operate their prisons over the next five years.

3. The primary reason for the dramatic increase in prison populations is the 'war on drugs,' which is not only increasing the number of prison admissions but is also increasing the rate of parole violations.

4. The already disproportionate rate of blacks and Hispanics being sent to prison will increase considerably, principally because of the war on drugs.[6]

Is the current war on drugs, with its emphasis on incarceration, an effective strategy for combating drug abuse in our society? The American Bar Association (ABA), in a recent report by its Ad Hoc Committee on Drugs, concluded that mandatory minimum sentences require expenditures "disproportionate to any deterrent or rehabilitative effect they might have."[16] The report stated the following:

> Even though costly, incarcerating large numbers of drug offenders might have some benefits if stiffer prison sentences incrementally reduced criminal behavior. There is no proof that mandatory minimum sentences have that effect. Moreover, when increased numbers of offenders serve increased time in prison, correctional facilities must devote more of their resources to providing minimal "warehousing" services, rather than rehabilitative services for fewer inmates. Accordingly, drug offenders who might benefit from drug treatment programs (and education and job training programs) do not have access to such programs.[16]

Health Status of Incarcerated Persons

Generally, American inmates come from lower socioeconomic strata. Thus, they generally have not had adequate health care on the outside and they tend to be at high risk for diseases such as AIDS, tuberculosis, hepatitis, heart disease, hypertension, and renal failure. In addition, many inmates are mentally ill or mentally retarded. Some of the more pressing health problems are noted below.

HIV Infection and AIDS.—The incidence of AIDS is 14 times higher in state and federal correctional systems (202 cases per 100,000) than in the U.S. population (14.65 cases per 100,000).[17] The higher incidence in corrections is due to the over-representation of persons with histories of high-risk behavior, especially intravenous drug use.

The National Commission on AIDS, in a 1990 report, found that the policy of mandatory sentencing for drug offenders (who have high rates of HIV infection) had placed an enormous burden on an already weak prison health care system. The Commission noted the following:

> Persons using injection drugs accounted for 28% of all adult and adolescent AIDS cases reported in the United States from 1981 through 1989. By choosing mass imprisonment as the federal and state governments' response to the use of drugs, we have created a de facto policy of incarcerating more individuals with HIV infection. Under the present policy, the percentage of drug offenders in the federal prison system will rise by 1995 from 47% to 70%. Clearly, we are thus concentrating the HIV disease problem in our prisons and must take immediate action to deal with it more effectively.[18]

According to the National Institute of Justice, as of November 1989, a total of 5,411 confirmed cases of AIDS were reported by state and federal correctional systems and 30 large jail systems.[17] Although the distribution of cases of AIDS was uneven—14% of the systems contributed 84% of the cases in 1989—only five systems reported having no cases by 1989; five years earlier, 26 systems reported having no cases of AIDS. The 1989 data revealed that for the first time, "the percent increase in cumulative total correctional cases in the United States (72%) exceeded the increases in cases in the U.S. population at large (50%)."[17]

It is estimated that many more inmates are HIV seropositive. In states conducting blinded epidemiologic studies, rates of HIV seropositivity ranged from 0.6% (Oregon and Wisconsin) to as high as 17% (New York).[17] Clearly, all correctional systems will have to confront the problems of HIV infection and AIDS. Dealing with the problem of HIV infection and AIDS will have a tremendous effect on the costs of correctional health care and must be considered when calculating future budgets. The lifetime cost of caring for a single person with AIDS is estimated at over $85,000 ($32,000 annually) and the yearly cost of caring for an asymptomatic HIV-infected person is over $5,000.[19]

Tuberculosis.—Tuberculosis, a disease once thought to be well controlled in the United States, is on the rise. The increased incidence is attributable, in part, to the epidemic spread of HIV infection. The incidence of tuberculosis in persons with AIDS is almost 500 times that of the general population, and the risk for developing active tuberculosis among HIV-infected persons who have positive tuberculin skin tests is estimated to be 8% per year.[20] The Centers for Disease Control (CDC) estimates that the incidence of tuberculosis among incarcerated persons in 1984 and 1985 was more than three times the rate in the general population. In 1987 in New Jersey, the rate (109.9 per 100,000) was 11 times higher than in the state's general population. In California, the rate (80.3 per 100,000) was six times higher. In New York prisons, cases of tuberculosis among inmates increased from 15.4 to 105.5 per 100,000 from 1976 to 1986, with 56% of cases occurring in HIV-positive inmates.[21] The magnitude of future problems with tuberculosis in patients with HIV infection depends on the extent of overlap between these populations.[20] Correctional facilities, with large numbers of intravenous drug abusers in crowded conditions, provides a dangerously good milieu for such overlap.

The CDC notes that transmission of tuberculosis in correctional facilities presents a health problem for both the institutions and the community into which inmates are released. In its guidelines for the prevention and control of tuberculosis, the CDC states the following:

> Control of TB is essential in correctional health care. Each correctional institution should designate an appropriately trained official responsible for operating a TB prevention and control program in the institution. A multiinstitutional system should have a qualified official and unit to oversee TB-control activities throughout the system ... the basic activities to be followed are surveillance, containment, and assessment.[22]

Of particular concern are recent outbreaks of multidrug-resistant tuberculosis in prison systems. The New York State system reported an outbreak that has resulted in the deaths of 14 persons, including 13 inmates and one correctional officer, since December 1990. Because of this outbreak, New York just instituted mandatory, annual tuberculosis screening for all inmates and correctional staff.[23]

Health Needs of Female Inmates.—The number of women in state and federal prisons more than tripled between 1980 and 1990 to an all-time high of 43,845.[3] Sixty percent are nonwhite women. More than three fourths of these women have children.[13] Some are pregnant when they enter prison or jail, although the precise percentage is unknown. These women often have high-risk pregnancies, despite their relatively young age (almost three fourths are between 18 and 34 years of age[13]). The lack of adequate prenatal care on the outside, coupled with extensive substance abuse and a higher incidence of chronic diseases (such as hypertension and diabetes) and communicable diseases (such as sexually transmitted diseases and AIDS), complicate their pregnancy management.

Women in correctional facilities have extensive health needs that few institutions are set up to meet. An estimated 41% of women in prison report previous sexual or physical abuse in their lives;[13] these include 23% that report abuse occurring before 18 years of age. These women carry with them the emotional and physical scars of this abuse.

The National Commission on AIDS took note of the general neglect of women's health care in correctional facilities, as well as the specific lack of services for HIV-positive women. The Commission reported the following:

> The lack of services for women and adolescents living with HIV disease is another area where the weaknesses in the correctional facilities parallel or exceed those in the general community.... In studies of the prison community, HIV seroprevalence rates among prison entrants are higher for women than for men. Clearly, this demands particular attention. Further, a significant number of women give birth to children shortly before they begin to serve prison sentences, or are pregnant and give birth during their incarceration. These women need prenatal services where confidential HIV testing is offered upon request. Special care to provide education and counseling is essential.[18]

Mental Disorders.—Studies estimate that between 6% and 14% of the correctional population may have major psychiatric disorders.[24] More than 13% of jail inmates in 1989 said that they had taken prescribed medication for an emotional or mental problem, and 8% indicated they had previously been sent to a mental hospital or treatment program by a court.[14] Mentally ill inmates are considered special-needs offenders and often require specialized housing and security in addition to such mental health services as psychological screening, counseling, psychotherapy, and medication.[24]

In a 1991 study, the General Accounting Office (GAO) found that 5.5% of all federal inmates had diagnosed Axis I mental disorders—not including those inmates whose disorder mainly involved use of drugs or alcohol.[25] Only two thirds of these inmates were involved in the facilities' psychology or mental health programs. In addition, two thirds of all Bureau of Prisons facilities reported having inmates with undiagnosed Axis I disorders, having inmates with diagnosed Axis I disorders who were not enrolled in treatment programs, or having inmates who were not receiving needed inpatient psychiatric care. More than half of all facilities reported that some mentally ill inmates went untreated because of insufficient resources (staff, programs, or facilities).[25] A 1990 advisory group put together to identify mental health care issues proposed extensive recommendations to improve the Bureau's identification management and delivery of such care, and some of these recommendations have been implemented.

Acute and chronic mental disorders in correctional settings can have dire consequences, including suicide, which is especially a problem in jails and detention centers. In 1990, suicide accounted for 30% of all inmate deaths in larger (more than 100 inmates) jails.[4] In a 1986 study, researchers identified 401 suicides in holding and detention centers and estimated that the suicide rate in detention facilities is approximately nine times greater than in the general population.[26]

Additionally, a smaller percentage of inmates is classified as mentally retarded or developmentally disabled, and these inmates require support services from the mental health program. A survey of state and federal correctional systems by McCarthy[27] showed that about 2.5% of the total inmate population was classified as mentally retarded, but other studies suggest that 10% may be a more accurate figure.[28]

Substance Abuse

According to a 1991 study by the GAO, nearly three quarters of all state inmates[29] and two thirds of all federal inmates[30] have substance abuse problems requiring treatment. In a recent Bureau of Justice Statistics survey, 58% of all jail inmates in 1989 reported regular drug use.[15]

Few offenders receive any treatment or services for substance abuse while incarcerated. The Bureau of Justice Statistics noted that about 5% of jail inmates were receiving treatment for drug abuse at the time of the survey. In a 1989-1990 American Jail Association survey, fewer than 20% of jails reported drug treatment programs involving paid staff (American Jail Association, personal communication). Three quarters of all jails did not provide group therapy, drug education, transition planning, and referral to community drug treatment agencies.

In state prisons, the GAO found that fewer than 20% of inmates with substance abuse problems were receiving any type of drug treatment[29] The GAO noted the challenges that states face in expanding treatment programs; these include 1) limited funding for providing treatment services; 2) security considerations; and 3) difficulties in assuring the availability of aftercare.

The GAO stated that only about 1% of the estimated 27,000 inmates in federal prisons who have moderate-to-severe substance abuse problems were receiving intensive treatment. Although the Bureau of Prisons has an ambitious plan to ensure drug treatment for all federal inmates in need, the GAO found that "implementation falls far short of meeting the needs of federal inmates."[30]

The extent of substance abuse among incarcerated persons and the need for treatment in this population have major budgetary implications for correctional institutions. The Bureau of Prisons estimates that the average cost of treatment in intensive programs is about $5,000 yearly per federal inmate.[30] This is in addition to the $18,000 average annual cost of incarceration in federal prisons in 1990.[30]

Resource Needs

Although it is possible to discuss the average cost of treating specific conditions that are prevalent in the inmate population, it is impossible to estimate, with precision, the total amount of funds needed to provide adequate health care in each institution. The costs of health care vary with the services supplied, the numbers and kinds of health care personnel, and with the nature of the delivery system. In the absence of this level of detailed information, we will need to look at broad numbers and use some proxy measures to determine the adequacy of correctional health care budgets.

The Cost of Correctional Health Care.—The cost of providing health services to inmates is influenced by factors within the criminal justice system and within medicine as a whole. In general, the cost of health services in the United States has escalated dramatically in recent years. According to the Health Care Financing Administration (HCFA), U.S. health care spending totalled $604 billion in 1989, an 11% increase in one year. The HCFA estimates that the factors leading to the increase in personal health care expenditures include general inflation (44% of the increase), industry-specific inflation (21%), and population changes (9%). The remaining increase is accounted for by other factors, including changes in technology, medical practice, and service intensity.[31]

The costs of correctional health care have escalated for many of the same reasons. In addition, litigation has resulted in court-ordered standards of care in many correctional systems, which forced those systems to increase health care spending to meet community standards of care.

In 1990, the National Commission on Correctional Health Care (NCCHC) surveyed the 50 state correctional systems and the Federal Bureau of Prisons to determine how much each was spending on health services.[8] The results showed not only an increase in the total dollars expended for health care, which one would expect from the population explosion, but also dramatic increases in the average cost per inmate.

On an average basis, state correctional systems increased their per inmate annual health expenditure 104% in seven years. They spent $906 per inmate for health care in 1982, $1,394 in 1985, and $1,848 in 1989.[8] If we estimate that half of these inmates have drug problems requiring intensive treatment, at an average annual cost of $5,000 (by Bureau of Prisons estimates), we find that states should be spending $2,500 per inmate on drug treatment alone. Although these cost estimates should be used with caution, it is clear that the magnitude of inmate health problems—including not only the target conditions we mention here but also the acute and chronic illnesses to which we are all susceptible—far exceeds current budgetary outlays for prison health care.

Problems and Costs in the National Drug Control Strategy Budget.—By its own report, the NDCS devotes 70% of the federal resources in combating drug abuse to law enforcement ("supply reduction") and 30% to prevention and treatment initiatives ("demand reduction").[7] Although the NDCS emphasizes incarceration, its budget does not reflect appropriate increases in operating costs such as health care. Its goal, through construction of new prison beds, is to reduce the federal prison system's overcrowding rate to 139% of rated capacity by 1995. This goal would be reached after construction of more than 50,000 beds at a total cost of more than $2.1 billion in the 1990 to 1992 period.[7] In comparison, the three-year total amount dedicated to drug treatment resources in federal prisons is $39 million. No mention is made of other resources for health care.

The NDCS notes that 92% of all incarcerated persons are confined in state and local prisons and jails and that these localities are principally responsible for handling most of the population expansion. Other than a challenge to state and local governments to explore ways to reduce operating costs and to provide drug treatment, the NDCS offers few solutions to the fiscal burdens imposed on localities by a policy of putting more drug offenders behind bars. As the ABA states, "Given the current state of the nation's correctional system, sentencing policies that are not based on realistic estimates of prison capacity or accompanied by additional resources to meet the demands are fiscally irresponsible."[15] We believe these budgetary problems threaten to overwhelm correctional systems and may doom inmates to unacceptable, substandard care.

The courts have affirmed that lack of resources cannot excuse systemic medical deficiencies, including shortages of qualified medical personnel, equipment or supplies.[32] "Thus, lack of funds ... does not justify operating a prison in an unconstitutional manner."[33] Incarcerated persons must receive health care that meets the currently accepted standard of care in the community, and adequate resources must be dedicated to providing that care. No less can be tolerated—ethically or legally.

Summary and Conclusions

Clearly, mandatory sentencing practices and the NDCS have overwhelmed correctional facilities to the point of crisis, without substantially alleviating the national problem of drug abuse or drug-related crime. As health professionals, we are seeing the human cost of putting more and more people behind bars. Funding for correctional health care has lagged far behind the explosive population growth. These policies have resulted in severe overcrowding, insufficient treatment programs for substance abusers, and increased prevalence of communicable diseases, particularly those associated with intravenous drug use. This is a national public health problem. Inadequate health services mean that inmates—nearly 10 million of whom are released each year from prisons and jails—may be returned to the community with diseases that threaten the health of the community. In other words, this affects us all.

In light of the above, we conclude the following:

1. Given its enormous human and financial cost, the NDCS, with its emphasis on incarceration, must be reconsidered. Its effectiveness in managing and preventing drug abuse must be evaluated, and alternative approaches that put less emphasis on criminal sanctions and more on prevention and treatment must be considered.

2. Correctional health care budgets must reflect the growing mental and physical health needs of the inmate population. Federal, state, and local officials must increase funding to provide comprehensive, integrated services to treat substance abuse, as well as conditions linked

to intravenous drug use—such as HIV infection and AIDS and hepatitis B infection. Systemic deficiencies in meeting the health needs of female inmates and of mentally ill inmates must be corrected.

3. Correctional health care must be recognized as an integral part of the public health sector. State and local health departments must join with correctional health professionals to control the spread of infection within corrections and to the public. This will necessitate additional resources for most health departments and/or coordinating agencies, which are already reeling from the effects of AIDS and the resurgence of communicable diseases such as tuberculosis.

4. All correctional facilities must implement and maintain standards, such as those for NCCHC accreditation, as one way to ensure minimally adequate health care delivery systems.

5. To respond to new and increasing demands, correctional care must evolve from its present reactive "sick call" model into a proactive system that emphasizes screening, early disease detection and treatment, health promotion, and disease prevention. We call for increased cooperation among academic institutions, state and local health departments, and departments of corrections to foster this evolution. This approach must be accompanied by resources for research that more accurately defines inmate health needs and explores how systems can more adequately meet these needs.

Appendix

Organizations

1. American College of Physicians (ACP): a professional association formed in 1915 that represents over 75,000 physicians whose specialty is internal medicine. The ACP was founded in 1915 to uphold high standards in medical ethics, education, research, and practice, with the overarching goal of ensuring the quality of patient care.

2. American Correctional Health Services Association (ACHSA): a professional association formed in 1975 that represents about 1,500 correctional health professionals from various disciplines (for example, medicine, nursing, pharmacy).

3. National Commission on Correctional Health Care (NCCHC): a not-for-profit 501(c)(3) organization formed in 1983 whose board of directors comprises individuals named by 32 professional associations including the American Medical Association, the American Bar Association, the American Nurses Association, ACP, and ACHSA. The NCCHC serves as the primary accrediting body for correctional health systems in the United States.

Terms

1. Jail: a facility operated by a local government (city or county) to detain persons before trial or to hold persons convicted of a misdemeanor who generally have a sentence of less than one year. Jails are also currently used to hold convicted felons awaiting transfer to a prison.

2. Prison: a facility operated by the state or federal government to confine convicted felons who generally have been sentenced to one year or more.

3. Correctional facilities: in this article, a term referring inclusively to both prisons and jails.

4. Juvenile institutions: a facility for holding detained or adjudicated youth who generally are less than 18 years of age.

5. Mandatory sentence: refers to a sentence in which incarceration is mandated by law.

6. Fixed sentences: refers to a sentence in which the length of time in a correctional facility is established by law. Many drug offenses carry both mandatory and fixed sentences as their penalty.

REFERENCES

1. Estelle *vs* Gamble, 429 U.S. 97 (1976).
2. Mauer M: Americans Behind Bars: A Comparison of International Rates of Incarceration. Washington, DC: The Sentencing Project; 1991.
3. Bureau of Justice Statistics: Prisoners in 1990. Washington, DC: U.S. Department of Justice; 1991.
4. Bureau of Justice Statistics: Jail Inmates, 1990. Washington, DC: U.S. Department of Justice; 1991.
5. Sourcebook of Criminal Justice Statistics, 1990. Washington, DC: U.S. Department of Justice; 1991.
6. Austin J, McVey AD: The 1989 NCCD Prison Population Forecast: The Impact of the War on Drugs. San Francisco, California: The National Council on Crime and Delinquency; 1989.
7. Office of National Drug Control Policy. National Drug Control Strategy. Washington DC: U.S. Government Printing Office; 1991.
8. Anno BJ: The cost of correctional health care: Results of a national survey. *Journal of Prison and Jail Health.* 1991; 2:105-34.
9. U.S. House of Representatives, Committee on the Judiciary, Subcommittee on Intellectual Property and Judicial Administration. Medical Care in the Federal Prisons: Hearing, 17 July 1991. Washington, DC: U.S. Government Printing Office; [In press].
10. Anno BJ: Prison Health Care: Guidelines for the Management of an Adequate Delivery System. Washington, DC; U.S. Department of Justice; 1992.
11. Office of Juvenile Justice and Delinquency Prevention. Children in Custody 1989. Washington, DC: U.S. Department of Justice; 1991.
12. Camp GM, Camp C: The Corrections Yearbook. South Salem, New York: Criminal Justice Institute; 1990.
13. Bureau of Justice Statistics. Women in Prison. Washington, DC: U.S. Department of Justice; 1991.
14. Bureau of Justice Statistics. Profile of Jail Inmates, 1989. Washington, DC: U.S. Department of Justice; 1991.
15. Bureau of Justice Statistics. Drugs and Jail Inmates, 1989. Washington, DC: U.S. Department of Justice; 1991.

16. American Bar Association. Responding to the Problem of Drug Abuse: Strategies for the Criminal Justice System. The Report of the Ad Hoc Committee of the Criminal Justice Section. Washington, DC; 1992.

17. Moini S, Hammett TM: 1989 Update: AIDS in Correctional Facilities. Washington, DC: National Institute of Justice; 1990.

18. National Commission on AIDS. HIV Disease in Correctional Institutions. Washington, D.C.; 1991.

19. Hellinger FJ: Forecasting the medical care costs of the HIV epidemic: 1991-1994. *Inquiry*. 1991; 28:213-25.

20. Barnes PF, Bloch AB, Davidson PT, Snider DE Jr: Tuberculosis in patients with human immunodeficiency virus infection. *N Engl J Med*. 1991; 324:1644-50.

21. Braun MM, Truman BI, Maguire B, DiFernando GT Jr, Wormser G, Broaddus R, et al: Increasing incidence of tuberculosis in a prison inmate population. Association with HIV infection. *JAMA*. 1989; 261:393-7.

22. Center for Disease Control. Prevention and control of tuberculosis in correctional institutions. *MMWR*. 1989; 38:18.

23. Mandatory Testing for TB in Prisons Begins with Guards. The New York Times. 19 Nov 1991; Sea B (column 3).

24. General Accounting Office. Mentally Ill Inmates: Better Data Would Help Determine Protection and Advocacy Needs. Washington, DC: General Accounting Office; 1991. GAO/GGD-91-35.

25. General Accounting Office. Mentally Ill Inmates: BOP Plans to Improve Screening and Care in Federal Prisons and Jails. Washington, DC: General Accounting Office; 1991. GAO/GGD-92-13.

26. Hayes LM, Rowan JR: National Study of Jail Suicides Seven Years Later. Alexandria, Virginia: National Center on Institutions and Alternatives; 1988.

27. McCarthy B: Mentally ill and mentally retarded offenders. In: Source Book on the Mentally Disordered Prisoner. Washington. DC: National Institute of Corrections; 1985.

28. Santamour MB: The Mentally Retarded Offender and Corrections Laurel, Maryland: American Correctional Association; 1989.

29. General Accounting Office. Drug Treatment: State Prisons Face Challenges in Providing Services. Washington, DC: General Accounting Office; 1991. GAO/HRD-91-128.

30. General Accounting Office. Drug Treatment: Despite New Strategy Few Federal Inmates Receive Treatment. Washington, DC: General Accounting Office; 1991. GAO/HRD-91-116.

31. Lazenby HC, Letsch SW. National health expenditures, 1989. *Heath Care Financ Rev*. 1990;12:i-26.

32. Newman *vs* State of Alabama, 503 F2d 1320 (1974).

33. Williams *vs* Edwards, 547 F2d 1206 (1977).

YOCON®

YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

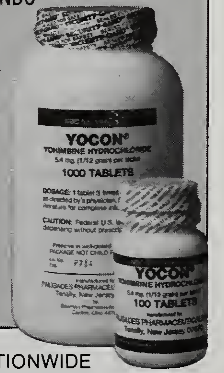
References:

1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85

AVAILABLE AT PHARMACIES NATIONWIDE

PALISADES
PHARMACEUTICALS, INC.
219 County Road
Tenafly, New Jersey 07670
(201) 569-8502
1-800-237-9083



DRUG INFORMATION UPDATE: HARTFORD HOSPITAL

Nitrate Tolerance—Mechanisms and Prevention

HEATHER KERTLAND, PHARM.D.

Introduction

NITRATE tolerance can be defined as the attenuation or loss of effect during chronic administration of the drug. Probably the first nitrate tolerance documented was in munitions workers. Workers complained of headaches upon first exposure to dynamite; these would slowly go away but would return after any absence, even as brief as a weekend. The recurrence could be avoided if some nitrate was placed on the band of their hat to provide continuous exposure while away from work. It was many years later that it was proposed that a patient may become tolerant to the clinically important hemodynamic and antiischemic effects of nitroglycerin. This article will review the evidence that tolerance exists, and discuss the possible mechanisms and ways to avoid it.

Evidence of Nitrate Tolerance

Exercise Performance.—The antianginal effects of nitrates are measured by an increase in the total exercise time or the time to significant angina occurrence during an exercise test. In all routes of delivery (oral, transdermal, and intravenous) an attenuation of such effects has been documented.

Thadani et al compared the effects of 15, 30, 60, and 120 mg of isosorbide dinitrate after a single dose on a treadmill exercise test and determined that there was a significant improvement for up to eight hours. After two weeks of therapy of 30 mg four times daily, the effects are significant for only two hours and greatly reduced from what had been seen with the single dose.[1] Using not only isosorbide dinitrate but also isosorbide mononitrate, other authors have demonstrated significant attenuation of effects after continuous exposure.[2,3,4] Similar findings are seen when transdermal systems of nitroglycerin are administered. When nitroglycerin patches (doses up to 80 mg/24 h) are applied, beneficial effects are seen for up to four hours after the first administration, but are not evident after 24 hours.[5] If the patch is changed every 24 hours and the effects measured at seven days, they remain attenuated.[6] Intravenous nitroglycerin that had been used experimentally in patients with chronic stable angina also showed similar results, that the effects were decreased to what they were at baseline after 24 hours of continuous infusion.

Hemodynamic Effects.—Attenuation of the hemodynamic effects is rapid, within 18 to 24 hours, with transdermal nitroglycerin use.[7] Attenuation of the beneficial effects on pulmonary capillary wedge pressure (PCWP) and mean arterial pressure can be seen when oral nitrates are dosed frequently, such as four times a day, or when nitroglycerin is administered as a continuous infusion.[8,9]

Mechanisms of Tolerance

There are three mechanisms proposed to explain the development of tolerance, excluding pharmacokinetic changes such as poor absorption or enhanced elimination.[10]

1. *Depletion of sulfhydryl cofactors.*—Organic nitrates enter into the vascular smooth muscle cell where they are metabolically activated to form nitric oxide or S-nitrosothiol. This process requires cysteine or a sulfhydryl group. S-nitrosothiol stimulates guanylate cyclase to convert guanosine triphosphate to cyclic guanosine monophosphate (cGMP). cGMP is responsible for a reduction in cytosolic calcium which leads to vasodilation (see Fig. 1). It is proposed that continuous nitrate therapy depletes

---

HEATHER KERTLAND, PHARM.D., Cardiovascular Therapeutics Fellow, Hartford Hospital; Assistant Clinical Professor, University of Connecticut, School of Pharmacy, Storrs.

This series on Drug Information Updates from Hartford Hospital are provided by the Drug Information Center, Department of Pharmacy Services, Hartford Hospital.

Editors: Moses Sing Sum Chow, Pharm.D. and Robert A. Quercia, M.S., R.Ph.

Editorial Board: D. Drezner, M.D.; G. Gousee, M.S.; R. Quintiliani, M.D.; R. Steven, M.D.; J. Laut, M.D.

the body's cysteine pool and therefore the nitrate is no longer able to activate the cascade of events described above.

2. *Activation of endogenous vasoconstrictor mechanisms.*— Nitrates may activate endogenous neurohumoral systems (specifically the renin-angiotensin-aldosterone system), resulting in sodium retention and vasoconstriction, thus limiting the effects of the nitrates.[11]

3. *Expansion of intravascular volume resulting in loss of hemodynamic efficacy.*—Nitrates may expand intravascular volume as demonstrated by a decrease in hematocrit. It is thought that there is a shift of water from the extravascular space to the intravascular space causing the increase in volume rather than any water retention occurring in the kidney.[10] This change in intravascular volume may negate any beneficial preload effect from the nitrate.

None of these theories alone can account for the changes seen with continuous administration of nitrates. It is probably a combination of effects that eventually results in nitrate tolerance.

Prevention of Nitrate Tolerance

As there are probably more than one component to nitrate tolerance several different methods have been proposed to avoid its development.

A) As nitrates need sulfhydryl groups donated from cysteine to have an effect, replacement of sulfhydryl donors may restore the lost effects. N-acetylcysteine (NAC) has been used as an exogenous source of cysteine and has been given to patients with congestive heart failure receiving nitroglycerin. Acutely, it reduced the amount of nitroglycerin needed to decrease the mean arterial pressure (MAP) and PCWP but this effect was not sustained at 24 hours.[12] The authors stated that NAC may enhance the effects of nitroglycerin,but does not prevent tolerance. Additional studies carried out in patients with chronic stable angina revealed that attenuation of effects could still be seen in patients who were receiving concomitant nitroglycerin and NAC.[8,13] Further work in this area is ongoing.

B) To counteract mechanism #2 (prevention of activation of neurohormones), angiotensin converting enzyme (ACE) inhibitors have been used to prevent the activation of neurohormones. In addition, if the ACE inhibitor captopril is used, it could act as a sulfhydryl donor since captopril has a sulfhydryl group. This hypothesis was assessed in 12 healthy volunteers. Both captopril and enalapril (does not contain sulfhydryl group) were able to prevent the attenuation of effects (as measured by a venodilator response) after 72 hours of continuous exposure to nitroglycerin.[14] But this needs to be verified in patients.

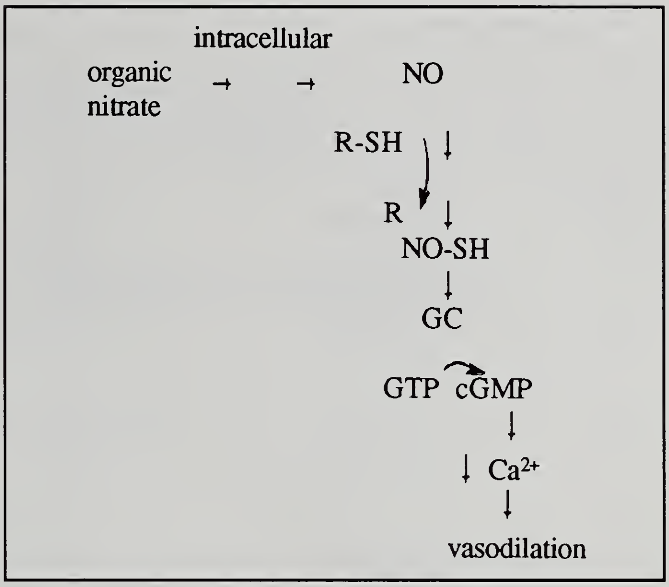


Figure 1.
NO=nitric oxide, R-SH=sulfhydryl donator, cGMP =cyclic guanosine monophosphate, GC=guanylate cyclase, GTP=guanosine triphospate

C) To modify the plasma expansion that may lead to nitrate tolerance, diuretics have been given in a double blinded fashion to healthy volunteers maintained on nitroglycerin patches. At the end of seven days there was no difference in the heart rate or blood pressure between the placebo or the diuretic treated group.[15]

D) The most effective method to date at preventing nitrate tolerance is a nitrate-free interval (see Table 1). Why the nitrate free-interval is effective at preventing the attenuation of nitroglycerin's effect has not been well established. When regular-release isosorbide dinitrate 30 mg is administered with a nitrate-free interval (ie, 7AM and 12PM or 7AM, 12AM, 5PM) it has been shown to result in a significant increase in exercise tolerance for up to five hours after the last dose compared to administering it without a nitrate-free period (7AM, 12PM, 5PM, and 10PM) where the effects are attenuated at two hours and beneficial effects could not be measured past this point.[3] Similarly with isosorbide mononitrate, eight hours of protection after the last dose has been documented when administered with a 17-hour nitrate-free interval (8AM and 3PM) compared to administering it with a shorter 12-hour nitrate-free period (10AM and 10PM) where again the effects are attenuated.[16] For most patients with stable angina pectoris no rebound nocturnal angina during the nitrate-free interval has been reported.

Table 1.

Regimens that Avoid Development of Nitrate Tolerance

Route	*Drug*	*Dose*	*Regimen*	*Reference*
po	isosorbide dinitrate	30 mg regular release	7AM, 12PM	#3
po	isosorbide dinitrate	30 mg regular release	7AM,12PM,5PM	#8
po	isosorbide mononitrate	20 mg regular release	8AM,3PM	#16
td	nitroglycerin patch	10-20 mg/24h	12h nitrate-free interval	#17,18

td—transdermal

Summary

Nitrate tolerance is of significant clinical concern and every effort should be made to avoid this undesirable effect. The mechanism of tolerance is not clear but probably is multifactorial. Although ACE inhibitors, diuretics, and NAC have been tried as preventative measures, the only method proven to maintain nitrate effectiveness in patients is a nitrate-free interval. The results of further studies designed to determine the actual period of time that the patient is protected will be helpful. This may guide us in choosing the optimal nitrate therapy.

REFERENCES

1. Thadani U, Fung HL, Drake AC, Parker JO: Oral isosorbide dinitrate in angina pectoris: Comparison of duration of action and dose response relation during acute and sustained therapy. *Am J Cardiol* 1982; 49:411-9.
2. Rudolph W, Dirschinger J, Reiniger G, Beyerle A, Hall D: When does nitrate tolerance develop? What dosages and intervals are necessary to ensure maintained effectiveness. *Eur Heart J* 1988; 9 (suppl A):63-72.
3. Parker JO, Farrell B, Lahey KA, Moe G: Effect of intervals between doses on the development of tolerance to isosorbide dinitrate. *N Engl J Med* 1987; 316:1440-4.
4. Kohli RS, Rodrigues EA, Kardash MM, Whittington JR, Raftery EB: Acute and sustained effects of isosorbide 5-mononitrate in stable angina pectoris. *Am J Cardiol* 1986; 58:727-31.
5. Thadani U, Hamilton SF, Olson E., et al: Transdermal nitroglycerin patches in angina pectoris. *Ann Intern Med* 1986; 105:485-92.
6. Parker JO, Fung HL: Transdermal nitroglycerin patches; duration of effect, and rapid tolerance. *Am J Cardiol* 1984; 54:1
7. Jordan RA, Seth L, Casebolt P, Hayes MJ, Wilen MM, Franciosa J: Rapidly developing tolerance to transdermal nitroglycerin in congestive heart failure. *Ann Intern Med* 1986; 104:295-8.
8. Packer M, Lee WH, Kessler PD, Gottlieb SS, Medina N, Yushak M: Prevention and reversal of nitroglycerin tolerance in patients with congestive heart failure. *N Engl J Med* 1987; 317:799-804.
9. Elkayam U, Kulick D, McIntosh N, Roth A, Hsueh W, Rahimtoola SH: Incidence of early tolerance to hemodynamic effects of continuous infusion of nitroglycerin in patients with coronary artery disease and heart failure. *Circulation* 1987; 76:577-84.
10. Packer M.: What causes tolerance to nitroglycerin? The 100-year old mystery continues. *J Am Coll Cardiol* 1990; 16:932-5.
11. Parker JO, Parker JD: Neurohormonal activation during nitrate therapy: A possible mechanism for tolerance. *Am J Cardiol* 1992; 70:93B-97B.
12. Dupuis J, Lalond G, Lemieux R, Rouleau JL: Tolerance to intravenous nitroglycerin in patients with congestive heart failure: Role of increased intravascular volume, neurohumoral activation, and lack of prevention with n-acetylcysteine. *J Am Coll Cardiol* 1990; 16:923-31.
13. Parker JO, Farrell B, Lahey KA, Rose BF: Nitrate tolerance: The lack of effect of n-acetylcysteine. *Circulation* 1987; 76:572-6.
14. Katz RJ, Levy WS, Buff L, Wasserman AG: Prevention of nitrate tolerance with angiotension converting enzyme inhibitors. *Circulation* 1991; 83:1271-7.
15. Parker JD, Farrell B, Fenton T, Parker JO: Effects of diuretic therapy on the development of tolerance during continuous therapy with nitroglycerin. *J Am Coll Cardiol* 1992; 20:616-20.
16. deBelder MA, Schneeweiss A, Camm A: Evaluation of the efficacy and duration of action of isosorbide mononitrate on angina pectoris. *Am J Cardiol* 1990; 65:6J-8J.
17. Thadani U, Bittar N: Effects of 8:00AM and 2:00PM doses of isosorbide 5-mononitrate during twice-daily therapy in stable angina pectoris. *Am J Cardiol* 1992; 70:286-92.
18. Demots H, Glasser SD: Intermittent transdermal nitroglycerin therapy in treatment of chronic stable angina. *J Am Coll Cardiol* 1989; 13:786-95.
19. Cowan TC, Bourke JD, Reid DS, Julian DG: Prevention of tolerance to nitroglycerin patches by overnight removal. *Am J Cardiol* 1987; 60:271-5.

Editorial

Rising Health Care Costs: Disease or Symptom?

TIMOTHY B. NORBECK

AS the health care reform debate intensifies, it is becoming more apparent that critics are pointing to physicians as the primary culprits for rising health-care costs. If the Task Force led by Hillary Rodham Clinton believes such charges and decides to frame the medical profession as the scapegoat for our system's ills, most assuredly we will not accomplish the important goal of containing the growth of health-care costs. Yes, physicians generally "do well by doing good," and yes, their decisions on behalf of their patients do substantially impact medical care expenditures as a whole, from prescriptions and splints to bypasses and transplants.

But most Americans don't realize that the physician's share of the health care dollar is about 19%—and has been about the same, incidentally, since 1950—but over half of that goes to overhead and administrative costs. What's left is subject to income tax, of course. Therefore, if one slashed physicians' before-tax incomes by 20% as some unknowing critics have suggested, the savings achieved would be less than 2% of total health care costs. Lost on the public is the fact that physicians' fees are not physicians' incomes, but also include part of the incomes of nurses, technicians, clerks, suppliers, landlords, etc. Also lost to most observers are the 65 hours a week that constitute most physicians' workweeks. At the minimum, they were at least 48% longer than the typical 40-hour workweek.

Additionally, the average age of a medical student graduate is 28. By the time residency programs are completed, most physicians actually begin their careers in their mid-thirties and carry a considerable debt from their medical school years. Any successful health reform enacted must address the root causes of illness (and their attendant expenditures) rather than the symptoms. An estimated 90% of lung cancer cases are caused by smoking. Capping budgets in treating those patients may save a few dollars but do virtually nothing to prevent the disease; likewise for cirrhosis, or other fatal diseases closely related to personal behavior. Smoking costs 400,000 lives a year along with $65 billion in health care costs and lost productivity. Alcohol and substance abuse add another $110 billion. Violent crime in our society, the homicide capital of the world, adds significantly more billions to those costs.

Our nation also has the dubious distinction of leading the civilized world in the number of teen pregnancies and low-birth-weight infants (two major factors in infant mortality). Just one of those consequences is that 90% of the men in prison today reportedly are the children of teen mothers. Thanks to U.S. technology, 20-ounce premature infants can be saved at a cost of $2,500 a day and up to $1.2 million for a 150 to 160 day stay in a neonatal unit. Other countries which might occasionally encounter such "preemies" don't incur such costs because the technology is not made available, and heroic efforts won't be made to save them.

The federal government has acknowledged in studies over the years that 50% of our mortality and health-care costs are attributable to accidents and illnesses relating to life-style abuses. One of the major reasons why we lead the world in per capita health expenditures is that our social problems also outweigh those in other nations. Another reason is our reliance on and expectations of the best technology that money can buy. A Harris poll indicated that 91% of the American public want the best possible health care available and 71% believe that health insurance should cover anything to save a life. Imagine the cost of providing a New Jersey woman with seven artificial joints—two shoulders, two hips, two knees, and one ankle. She actually danced at her daughter's wedding! Who can place a price tag on her new quality of life?

As submitted to the *Hartford Courant*, and published Sunday, 28 March 1993.

TIMOTHY B. NORBECK, Executive Director, Connecticut State Medical Society.

Some health-care costs are going to rise regardless of what we do. Our Medicare enrollees increase by 700,000 each year, and our over-85 population is the fastest growing segment of our population—increasing at a rate three times that of the population at large. We must acknowledge this happy achievement, but as one might expect, their health-care expenditures are six times higher than for those under 65, and 2-1/2 times higher than for a 65 year old. Entertainer George Burns recently said that "one of the nice things about being 90 is that there's less peer pressure." That may be so, but in fact there are many who have left him behind. Some 37,000 Americans have reached their 100th birthday, twice the number just 10 years ago. Related to these important demographics is the need for long-term-care, a pressing and expensive issue which must be addressed. Americans at age 65 have a 40% chance of spending some time in a nursing home and a 23% chance of being there for at least a year, and these costs devastate so many of elderly and their families.

Catastrophic illnesses are still another major factor in rising health care costs. Only one percent of us account for 29% of total health care expenditures, and 5% are responsible for as much as 50%. Only 18.8% of Medicare beneficiaries receive 80% the program's total payments. Some 24% of those costs are attributable to chronic illnesses associated with heart disease, cancer and stroke. As one can observe, it will not be easy to cut such expenditures without adversely affecting the patient's outcome. Thanks to such effective medicine and surgery, our average life expectancy is again rising at unprecedented rates.

For all its sophistication and technological resources, however, our health-care system suffers from one costly low-tech malady—unnecessary paperwork. There are about four billion medical claims each year which appear on as many as 1,500 different claim forms. Needless administrative costs must be curbed. Physicians are interrupted in their work by calls from "managed-care" reviewers, some of whom can't even spell the disease or pronounce the procedure. Other similar "hassle factors" are causing physicians to leave the practice of medicine early. One recently complained: "I've had it with these third parties second-guessing my first-rate care." In the past two years, the government has imposed complex new coding procedures, the Resource Based Relative Value Scale (RBRVS), the Clinical Laboratory Improvement Act (CLIA) regulations, the Occupational Safety and Health Administration (OSHA) standards and the Americans with Disabilities Act (ADA) requirements, all of which have substantially increased physicians' administrative costs. And yet today, government officials talk of how physicians will be expected to do more for less.

Some people believe that the government should play a greater role in whatever new health reform is enacted. While it is true that health-care costs have increased by 1,200% since the mid-60s when Medicare and Medicaid were introduced, federal government health-care expenditures over that period escalated by 1,800% Another federally-administered remedy would only serve to exacerbate the problem.

Few question the need for health-system reform. The 35 million uninsured Americans must have coverage so they can receive appropriate preventive care rather than be carried into the hospital emergency rooms *in extremis*. But we must recognize and accept that the costs of such insurance will add many new billions of dollars. It will also be important to contain the present growth of health care costs rationally. Physicians point to the potential of practice parameters, outcomes research and technology assessment for future savings. A great majority of the public supports tort reform as another means of achieving savings in our health-care system. The costs of "defensive medicine," that is, the tests and procedures undertaken in order to avoid exposure to a malpractice suit, are estimated to run anywhere between $25 billion and $125 billion a year.

> *It doesn't serve any useful purpose to blame any one sector for escalating health-care costs. Society's problems require societal solutions.*

To their credit, Hillary Rodham Clinton and President Clinton recognize that individual responsibility plays an important role in rising health-care costs and have urged Americans to improve their life-styles. In addition, we must as a nation address our social problems which influence the quality of life for so many of our citizens as well as increase the health-care expenditures of those who pay the bill.

A sound commitment to addressing poverty, smoking, alcohol and substance abuse, ignorance, poor housing, joblessness, violence, and self-destructive life-styles will do more to save our system's health-care dollars than any other measures we can take.

The Clintons and the Congress have an immensely difficult job ahead of them. It doesn't serve any useful purpose to blame any one sector for our escalating healthcare costs. Society's problems require societal solutions. How can caps or controls placed on medical services or payments successfully arrest expenditures when such limits cannot be placed on illness? Suggested by some as a panacea, price controls such as payment freezes will have little or no effect on the problem. Volume, not individual services, drives costs. For example, price controls have been in effect for 12 years as an integral part of the Medicare program. They haven't contained costs in any way, but they have exacerbated cost-shifting problems.

There is only one way to increase access, contain costs, and maintain the present high quality of services rendered: we must all accept some responsibility to improve our own health habits where we can, and expend the necessary resources to provide basic coverage to the uninsured and to eradicate our social problems.

As economist Herbert Stein once said: "America is a very rich country. We are not rich enough to do everything, but we are rich enough to do everything that is important." A rational approach to health reform is important to this country's future, and everything to this nation's health.

Lyme Disease

The Lyme Care Center seeks qualified physicians throughout Connecticut to whom patients presenting symptoms of early, middle stage and chronic Lyme disease can be referred for treatment. Candidates for referrals must treat aggressively, appropriately, and compassionately. To those physicians who qualify for inclusion in our network, we offer a tremendous opportunity for practice enhancement. **Interested physicians should contact Joseph A. Hernandez, Managing Director.**

1-800-842-5248

HoMed/The Lyme Care Center
2371 Boston Post Road
Guilford, CT. 06437-2315

HoMed

THE LYME CARE CENTER℠

C. CHARLES BURLINGAME, M.D.

JOHN C. NEMIAH, M.D.
has been honored as the recipient of the 1993
C. CHARLES BURLINGAME, M.D. AWARD
for his outstanding leadership
in psychiatric research and education

Past Recipients
1988 Robert Kellner, M.D., Ph.D.
1989 William T. Carpenter, Jr., M.D.
1990 Dennis P. Cantwell, M.D.
1991 George E. Vaillant, M.D.
1992 A. John Rush, M.D.

THE INSTITUTE OF LIVING

400 Washington Street, Hartford, CT 06106 • (203) 241-6900

"Filling out insurance forms gives my patients a pain in the neck."

—*Dr. Patrick J. Carolan, Orthopedics, Bridgeport, CT*

"Good medical care was once as easy as seeing your doctor. Complications set in. Forms to fill out. Deductibles. Hidden costs. Partial coverage. Non-medical intrusions into the patient-physician relationship.

"Doctors decided to do something about it. Through our State Medical Society, we first created **CSMS-IPA,** Connecticut's only *statewide* IPA. Then we launched **M.D. Health Plan,** based on a simple principle: The best medicine for *patients* is practiced by *physicians*— not insurance companies, not benefit managers, not cost analysts.

"It worked! Now the fastest growing health plan in Connecticut and the choice of over 68,000 patients, our company is 100% owned and 100% directed by practicing physicians. Why not?"

Patrick Carolan, M.D.,

M.D. HEALTH PLAN

6 DEVINE STREET • NORTH HAVEN, CT 06473 • (203) 776-5759 • 1-800-345-9272

50 Years Ago

From *The Connecticut State Medical Journal*

March 1943

Medicine and the War

Military Deaths in 1942

The Metropolitan Life Insurance Company, in its January 1943 Statistical Bulletin, reviews the military death toll in the various countries. Last year's losses are the greatest so far in the current World War and are estimated at about 2,000,000 soldiers and sailors. Among American fighting men the military losses for 1942 probably were at least 11,000 and possibly as many as 15,000. This uncertainty of figures is due to the lack of information to be obtained from the Japanese relative to the outcome of our men listed as missing. Our 1942 losses exceed all of those suffered by us in the first 15 or 16 months of the first World War, but by the end of last year we had three times as many men serving overseas as on the corresponding date of World War I.

The Japanese losses for 1942, including China, have been at least 100,000 and possibly over 125,000. Two operations alone—the engagements in the Macassar Straits in January and in the Solomons in November—cost Japan an estimated 40,000 to 70,000 troops by drowning. In China, Japan's estimated losses are nearly 40,000.

German losses are believed to have reached between 500,000 and 750,000 during the year 1942. In addition to the Russian losses which increased rapidly as the year progressed, Germany lost 40,000 to 50,000 soldiers and sailors killed in North Africa, over western Europe, in the Atlantic, and in occupied countries.

Russia apparently suffered the heaviest toll of all the belligerents last year. A conservative estimate places their military deaths at 750,000 to 900,000. This places them on a scale at least equal to those sustained in any year of World War I.

Rumania has suffered heaviest of the Axis satellites, probably losing 50,000 soldiers last year. Italy lost not more than 30,000 to 40,000 soldiers and sailors in 1942. Great Britain and the countries of the Empire suffered relatively few military deaths last year, probably not more than 30,000. The Chinese have probably lost 100,000, Yugoslavia many thousands, Free and Vichy French together not more than 1,000 to 2,000, and the Dutch a moderate number. Hungary has lost rather heavily, Bulgaria and Finland, on the other hand, relatively few.

From The Connecticut State Medical Journal, April 1943.

Pennsylvania Psychiatrist Talks on Shell Shock

According to Edward A. Strecker, professor and head of the department of psychiatry at the University of Pennsylvania in Philadelphia and president-elect of the American Psychiatric Association, the most common mental casualty in modern warfare is the "shell shock" of the newspapers, or war conversation hysteria.

Dr. Strecker spoke recently at the Neuro-Psychiatric Institute in Hartford. In World War I he held the rank of Maj or in the Medical Corps of the United States Army and was division neuro-psychiatrist for the 28th Division. His lecture on "Military Neuro-Psychiatric Disabilities and Their Treatment" on 4 March was the eighth in a series of twelve lectures on psychiatry and neurology sponsored by the Institute in its ninth annual series.

"War conversion hysteria or shell shock is an abnormal solution of a strong emotional conflict which takes place in the soldier between his ego-instinct (the instinct of self preservation) which is strongly moved by such emotions as fear, horror and revulsion, and the opposing claims of soldierly ideals and disciplinary reactions." In other words, when the conflict within the soldier, which is common among all of today's active combatants, becomes too strong, the soldier responds by a state of shell shock.

Dr. Strecker outlined methods of treating war conversion hysteria and pointed out that many of these mental casualties could be prevented through greater care in the selection of fighting men and through more thorough conditioning for battle among those who are selected.

"In these days every physician in or out of the armed services will have to practice psychiatry," Dr. Strecker continued, in stressing the tremendous effect which mental casualties have upon the morale of soldiers. Statistically and economically, the effects are also tremendous, he said.

"Psychiatry in the armed services, particularly under combat conditions will have to be 'rough and ready' but skillful," he concluded. "This is the acid test of psychiatry today."

INQUIRE TODAY.

ENDORSED BY
THE CONNECTICUT STATE MEDICAL SOCIETY
—OFFERING—

- Hospital, Surgical and Major Medical Insurance
- Term Life Insurance
- Employee Benefit Package

—FEATURING—

- OFFICE OVERHEAD AND DISABILITY INSURANCE PACKAGE

  [Rates based on a 30 day waiting period for ages 30-39]

  $1500.00 weekly disability indemnity
  semi-annual premium is $528.15*

  $20,000.00 monthly overhead indemnity
  semi-annual premium is $561.00

* Optional Residual and COLA benefits available

ayr

AYR INSURANCE AGENCY

105 SANFORD STREET HAMDEN, CT 06514 288-4302

THE PRESIDENT'S PAGE

Medicare Mess

"It is a function of government to invent philosophies to explain the demands of its own convenience," wrote the American journalist Murray Kempton in 1962. When Medicare was created it was hailed as a model for nationalized health and the solution for the ills of the society. But over the years the Medicare problem has evolved into a nightmarish bureaucratic mess. What was supposed to be good for the providers and recipients has become the battleground between them; it has made the doctor and the patient into antagonists. The executive branch and the congress and their agents, the HCFA, have written so many rules and regulations and established so many control mechanisms that the doctors of this country are opting out of the program. Medicare has frozen the fees for the past few years, and the increase in their payment methodology is not even sufficient to cover the overhead expenses and inflation. The RBRVS method, which was supposed to equalize the payment between the higher paid super specialists and generalists, has resulted in a decrease in payment to everyone and has caused divisions among medical and surgical specialties. The medical profession is not even able to handle the price gougers because of the Federal Trade Commission's frightening rules about price fixing and antitrust laws. Andrew Jackson in 1832 said: "There are not necessary evils in government. It's evils exist only in its abuses."

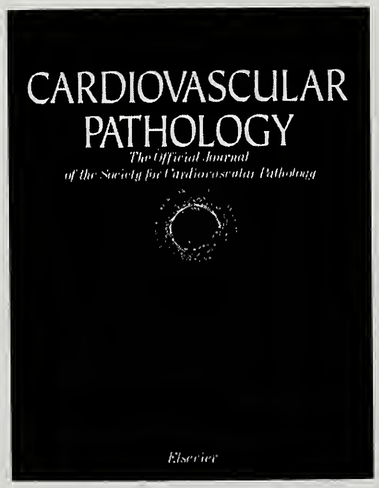
Adding insult to injury the federal government is planning to do more damage to the Medicare program, all in the name of cost containment. The following reductions may be forthcoming in the future budgets of the federal government:

1. Cap the Part B premium and limit the increase.
2. Reduce the payments to physicians.
3. Reduce practice expense relative value units or geographic practice cost index.
4. Reduce the physician's Medicare volume performance standards.
5. Bundle medicare payments for radiology, pathology, and anesthesia services into a fixed payment per discharge. Payment would be made to the hospital for distribution.
6. Reduce the surgical fees by the amount of any separate payment for an assistant.
7. Charge for any paper claims to encourage electronic billing.
8. Reduce payment for durable medical equipment.
9. Reduce payment to the hospital for indirect and direct medical education.

The federal government has done nothing to control the cost of products and supplies that are used by doctors and the hospitals. They have done nothing to rein in the soaring malpractice costs and the costs for defensive medicine practiced as a result of that threat. They have done nothing to control the cost of prescription and over-the-counter drugs. They have not allowed the elderly to opt out of the Medicare system completely and buy their own insurance in the open market with all the available HMOs and other provider networks. John F. Kennedy said: "The basis of effective government is public confidence." As far as the Medicare mess is concerned, nobody has any confidence in the system nor in the government that administers it. The Medicare recipients are burdened with so many rules and regulations and gobbledygook that they are miserable. The providers are subjected to layers upon layers of bureaucratic red tape and cost cutting so much so that it has become a losing proposition to treat the sick.

What can we physicians do in this atmosphere of distrust and antagonism? Civil disobedience comes to our minds. Civil disobedience as prescribed by Thoreau, Tolstoy, Gandhi, and King. This may cause alarm amongst our colleagues and dismay amongst our patients. It would impose a tremendous burden on the elderly. Some of the doctors are already not accepting new Medicare patients and others are seeing them without charge so that they can avoid the hassles. I hope that our government and its system will not drive us to these extremes. "Government is at best but an expedient," wrote Henry David Thoreau almost 150 years ago.

Sultan Ahamad, M.D.
President


CARDIOVASCULAR PATHOLOGY
The Official Journal of the Society for Cardiovascular Pathology
Elsevier

Essential reading for cardiologists and pathologists . . .

Editor-in-Chief:
Stephen M. Factor, MD,
Albert Einstein College of Medicine,
Bronx, New York, USA

Associate Editors:
Margaret E. Billingham, MD,
Stanford University Medical School,
Stanford, California, USA
Sherman Bloom, MD,
The University of Mississippi Medical
Center, Jackson, Mississippi, USA
Robert B. Jennings, MD,
Duke University Medical Center,
Durham, North Carolina, USA
Takeshi Shozawa, MD,
Akita University School of Medicine,
Akita, Japan
Gaetano Thiene, MD,
The University of Padua,
Padua, Italy

Send orders and sample copy requests to:

in North America
Elsevier Science Publishing Co., Inc.
Box 882, Madison Square Station
New York, New York 10159

outside North America
Elsevier Science Publishers
Box 211/1000 AE Amsterdam
The Netherlands

For faster service, call or fax today to place your order:

Tel. No.: (212) 633-3950
Fax No.: (212) 633-3990

NOW IN CURRENT CONTENTS!

CARDIOVASCULAR PATHOLOGY

Official Journal of the Society for Cardiovascular Pathology

Editor-in-Chief:
Stephen M. Factor, MD

Cardiovascular Pathology is a quarterly journal presenting articles on topics that cover the entire spectrum of the discipline. The Journal's primary objective is to publish papers on disease-oriented morphology and pathobiology from pathologists and non-pathologist investigators in the cardiovascular field.

Subjects range from vascular pathobiology to mechanical prostheses, from molecular biology to clinical cardiac disease, from experimental ischemia to descriptive gross morphology. **Cardiovascular Pathology** also publishes timely, informative reviews on scientific areas in cardiovascular pathology, and case reports that represent significant or unique observations.

Articles recently published in **Cardiovascular Pathology** include:

- Cardiac Transplant-Associated Vascular Disease: Evolution, Immunophenotype, and Evidence of Endothelial Activation, *K. Kottke-Marchant, W.E. Newman, and N.B. Ratliff*
- Pathogenesis of a Double Rupture (Septal and Free Wall Rupture) After Acute Myocardial Infarction, *R.S. Larson, E.J. Haskel, and J.E. Perez*
- Branch Location in the Left Anterior Descending Coronary Artery and Its Relation to the Known Distribution of Early Atherosclerosis, *N.R.B. Cary, T. Biggs, and W.A. Seed*
- Transcriptional Regulation of Calcium-Activated Neutral Protease in Cardiomyocytes of Hypertensive Rats, *M.N. Kumar, P. Grammas, F. Giacomelli, and J. Wiener*
- Intimal Thickening of Peripheral Pulmonary Vein: Analysis of 139 Consecutive Autopsy Cases, *M. Sageshima, H. Masuda, K. Kawamura, and T. Shozawa*
- Further Morphological Observations on Hearts With Twisted Atrioventricular Connections (Criss-Cross Hearts), *J.W. Seo, S.J. Yoo, S.Y. Ho, H.J. Lee, and R.H. Anderson*

Cardiovascular Pathology is abstracted in: Current Contents/Clinical Medicine; Current Contents/Life Sciences; and Current Awareness in Biological Sciences/CABS.

1993 Subscription Information
ISSN **1054-8807** Volume 2 (4 issues)
Institutional Rate: $145.00 Personal Rate: $85.00* Resident Rate: $68.00†
Subscribers outside the U.S., please add $23.00 for postage and handling.

†The Resident Rate is valid for two years only.
*The personal rate is available to individuals only. A home, hospital or medical school address must be used for delivery. Orders from non-U.S. customers must be prepaid.

3/93 XIMB

Doctors, Patients, and the End of Life

JOSEPH M. HEALEY, J.D.

IT is an ironic commentary on contemporary medicine that more Americans would recognize the name of Jack Kevorkian than would recognize the names of the most recent American winners of the Nobel Prize in Medicine. Dr. Kevorkian has succeeded in attracting widespread attention to our societal discomfort about what the role of a doctor should be during the dying process. By his participation in at least 15 deaths, Dr. Kevorkian has challenged the traditional ethical and legal prohibitions against physician involvement in acts that lead to the deaths of patients or in aiding or assisting patients to commit suicide. Our societal ambivalence about death contains within it a layer of ambivalence about allowing physicians too active a role in the deaths of their patients. The actions of Dr. Kevorkian have questioned whether the traditional distinctions between killing and letting die, between acts that lead to death and omissions that lead to death, distinctions that prohibit the former but legitimize the latter, are still desirable public policy. These questions will be the focus of this and the next column.

An initial stumbling block in attempts at clarifying these distinctions is the range of variables in such cases. The patient may be competent or incompetent; a willing, unwilling, or neutral participant and be located anywhere on the spectrum of health status from otherwise healthy to terminally ill. The action or the doctor may involve (1) withholding or withdrawing beneficial treatment; (2) withholding or withdrawing nonbeneficial treatment; (3) acts producing unintended but foreseeable consequences ("double effect"); or (4) acts intending the death of the patient. It is difficult to identify principles sufficiently flexible yet sufficiently clear to be useful in providing guidelines for the diverse cases likely to arise. A second barrier involves a growing recognition that traditional formulations of ethically and legally permissible conduct have limitations that undercut their usefulness. In a recent review article, Alan Meisel described the approach of courts in addressing such issues, affirming the traditional acceptance of actions construed as "letting die" and the traditional reluctance to permit actions construed as "killing." However, Meisel underscored the limits of this distinction:

> Even as a general proposition, the distinction that the courts have established is far too facile. There is just not the clear line between killing and letting die that many courts would like there to be. Even in the simplest case—for example, when a competent, terminally ill patient dies when a ventilator is withdrawn—the courts are wont to say that the patient was permitted to die (not killed) because his medical condition was such that he could not breathe without a ventilator, and that the cause of death was that same medical condition, rather than the removal of the ventilator.
>
> It may well be that as a matter of law, morals, and policy we do not wish to penalize the physician who honors the request of a competent patient to terminate life-sustaining treatment; indeed, we might wish to encourage such behavior as an indication of respect for patient autonomy. However, that does not, per se, make the physician's conduct into a "letting die" rather than a "killing" any more than does labeling it a killing make the conduct morally or legally wrongful,...Letting a patient die is morally and legally acceptable only under certain conditions, the most general of which is that the physician has no duty to continue to try to keep the patient alive....What makes such conduct acceptable, when it is, is not the fact that it is a letting die—indeed, whether it is an instance of one or the other is itself debatable—but that the physician is under no duty to continue to treat the patient because there has been valid consent by the patient, or by one authorized to speak for the patient, to the physician's conduct, alone or in combination with other factors (such as the patient's dim prognosis). And what makes such conduct wrongful, when it is, is that the physician has not been absolved of the duty to continue treatment by the same factors.[1]

The key issue is when such a duty exists, what its precise nature is, and how it should be carried out. Discussion of these questions will be continued in next month's column.

REFERENCE

1. Meisel A. The legal consensus about forgoing life-sustaining treatment: Its status and its prospects. *Kennedy Inst Ethics J*; 1992;2;327-8.

JOSEPH M. HEALEY, J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

Correspondents: Joseph M. Healey, J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington, CT 06030-1910.

WHO SAYS YOU CAN'T JUGGLE IT ALL ?

ProMed says you can. As a medical provider you strive to keep your patients healthy. As a businessman you aim to run an organized and efficient office. At ProMed, we can provide you with a medical office system to meet your most basic business needs — or we can provide you the advantages of a multi-user, multi-tasking operating system. And, as your practice grows, so can your ProMed office system to meet your changing needs.

With ProMed, dedicated service and comprehensive training are only the beginning:

- ☑ Faster and more accurate electronic claims submission
- ☑ Reduced paperwork
- ☑ Detailed financial and clinical management reporting
- ☑ Complete installation and support services

As the leading provider of medical office systems in Connecticut, ProMed saves time and money for more than 1,000 of the state's medical professionals.

You take care of your patients, we'll take care of the rest. For more information, call Bob Seekamp at ProMed: 1-800-922-3385.

ProMed
SYSTEMS INCORPORATED
221 Whitney Avenue
New Haven, Connecticut 06511

Health-Care Reform and Long-Term Care

ROBERT U. MASSEY, M.D.

LONG-TERM care insurance should be one of the components of health-care reform, but it may not even be close to the top of the priority list. Its costs are too high for more than a small percentage of the population, and could be reduced only by spreading premium payments, like Social Security, over a long period of time. But the most serious problem, according to Anne R. Somers, "...is a total 'disconnect' between the [long-term care] insurance policy and the provision of care, leaving even the expensively insured patient unprotected as to the quality of the covered services."[1]

A definitive history of the care of the elderly in America has yet to be written; the specialty of geriatrics is about where pediatrics was in the first decades of this century. The nursing home has evolved remarkably during my half century in medicine, but its quality has remained spotty, probably because most long-term care ventures began as businesses driven by profit rather than as community services. Tougher standards have improved quality, or at least the appearance of quality, but in the long run profitability is a poor objective when a community seeks to care for its dependent elderly members. Standards can be skirted, and regulation is uneven and expensive; the only assurance of quality is conscientious, dedicated administration, a committed, attentive board, and an intelligent, skillful, compassionate professional staff. Business-like, but not a business!

From a warehouse for the chronically ill, impoverished, or impaired elderly to a lively community working to meet the needs and fulfill the reasonable wants of older women and men is a long, but not impossible, step. Continuing care retirement communities (CCRCs) are one answer, but not everyone's solution, and still too costly for most. From assisted home living to hospice, with all levels in between, ought to be the objective of any long-term care plan, supported by some form of long-term care insurance.

> But regulation and social policy is often inconsistent and sometimes makes good care harder rather than easier to provide—in part because we have not, as a society, truly worked out a moral vision and a set of aspirations for what we want nursing homes to be.[2]

This is hardly surprising; we have had the problem for little more than 50 years. When most retirees lived only six months or a year after retirement; when families stayed put, and half of them lived in small towns or on the farm; when most wives and mothers were at home all day, helped by one or two of the older daughters, care for grandmother was an expected part of family life. Few worried about a moral vision of what nursing homes should be. There were county old people's homes for those few elderly who had outlived their families, or whose children had moved away, perhaps too poor or too shiftless to care for them. These were government homes, now comprising only 5% of the 19,000 nursing homes in the United States.[2]

Society rarely works out a clear moral vision of institutions or anything else, but thoughtful, far-seeing individuals dream dreams and build models, some of which work, and the successful models become society's vision and are then replicated. We are in that process now; CCRCs are one model. There are others: nursing homes that provide for assisted living, home care, day care, sheltered living, skilled nursing care that is but little removed from hospital care, especially now that hospitals have turned into vast intensive care units that are quite inappropriate for most geriatric care, and finally care at the very end of life, hospice care. They have yet, for the most part, failed to evolve from institutions to moral communities whose citizens help to run the place, but the best ones know that and are working on it.

The specialty of geriatrics and the institutions that we call nursing homes, although some are much more than that, are just beginning their history. To grow and mature to meet society's needs they will require close attention from those who are trying to figure out how to nurture our health-care system in the 21st century.

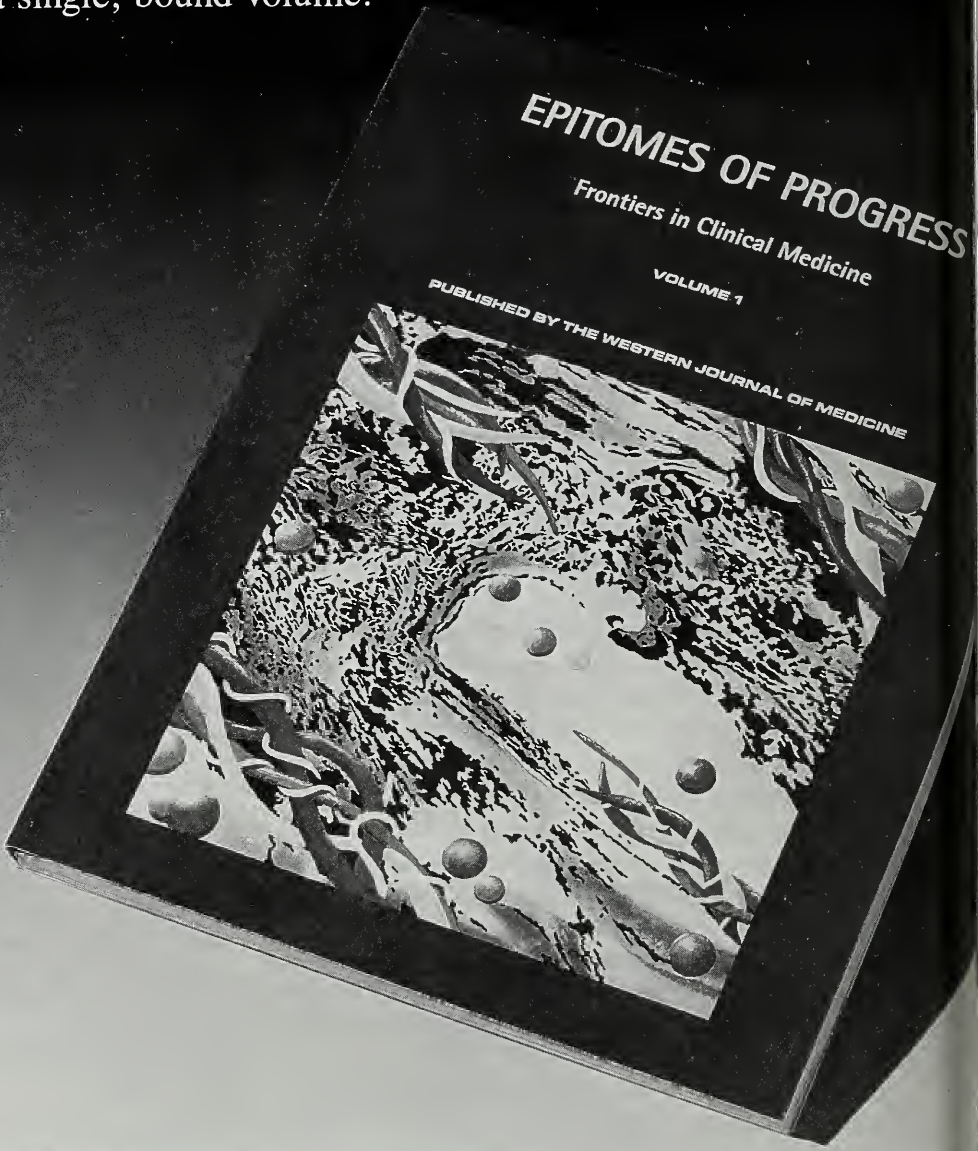
REFERENCES

1. Somers AR: "Lifecare": A viable option for long-term care for the elderly. *J Am Geriatr Soc* 1993; 41:188-91.
2. Collopy B, Boyle P, Jennings B: New directions in nursing home ethics. *Hastings Cent Rep* 1991, Special Supplement Mar-Apr.

ROBERT U. MASSEY, M.D., Professor Emeritus, Division of Humanistic Studies, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

epitome

i-ˈpit-ə-mē\ *n* [L, fr. Gk *epitomē*, fr. *epitemnein* to cut short, fr. *epi-* + *temnein* to cut — more at TOME] (1520) **1 a** : a summary of a written work **b** : a brief presentation or statement of something **2** : a typical or ideal example: EMBODIMENT (the British monarchy itself is the ~ of tradition — Richard Joseph) **3** : Plural — *Epit-o-mes* — A regular FEATURE of *The Western Journal of Medicine*, now available in a single, bound volume.


EPITOMES OF PROGRESS
Frontiers in Clinical Medicine
VOLUME 1
PUBLISHED BY THE WESTERN JOURNAL OF MEDICINE

Each set of **EPITOMES** is a concise and clear review of the important advances in a medical specialty, chosen and written by experts in that specialty. Now, for the first time, a special bound volume of **EPITOMES** is available. For only $19.95 you can have all the latest advances in 24 different specialties in one attractively designed volume. An excellent reference for your library. A useful gift. Hurry! Supplies are limited. Order a copy today.

YES, SEND A SPECIAL BOUND EDITION OF **EPITOMES** TO:

The Western Journal of Medicine

☐ Yes, send me ____ copy(ies) of the Epitomes book at $19.95 each.

☐ I want to receive Epitomes every month. Please enter my one-year subscription to *WJM* for only $40.00.

For additional information, call (415) 882-5179.
Please mail this coupon with enclosed payment to:
Circulation Department
P.O. Box 7602, San Francisco, California 94120-7602

Name ____________________________
Street ____________________________
City ____________________________
State ____________ Zip ____________
☐ Check enclosed Charge my: ☐ VISA ☐ MasterCard
Card Number ____________________________
Expiration Date ____________________________
Signature ____________________________

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgement), provided that the are submitted in duplicate, signed by all authors, typewritten in double spacing, and do not exceed 1-1/2 pages of text (excluding references). They should not duplicate similar material being submitted or published elsewhere. Letters referring to a recent Journal article should be received within six weeks of the article's publication.

SUDDEN CARDIAC DEATH

To the Editor: The event of Sudden Cardiac Death reflects a momentary failure in blood distribution. The neurogenic orchestration of vessel spasms and dilations to make the blood go only where function is needed is continuous. That correct motion of blood, a tiny percentage of the body's weight, sparks life. Vascular pathology is the loaded gun, emotion is the trigger pull of fatal dysfunction. Death is from the two events together. The Mystery of Life is dynamic coordinated motion!

Autopsy shows anatomic pathology, but not physiologic pathology; death is a defunct state. Medical science falls short in proving the cause of Sudden Cardiac Death.

Sidney Vernon, M.D.

Willimantic

IN RESPONSE TO DR. AHAMED

To the Editor: In the November issue Dr. Ahamed mentions that at his hospital a continuous communication takes place between physicians and administrators aimed at improving hospital-staff relations. Because such interactions are often unproductive, he should explain exactly how the ongoing dialogue occurs. For example, is it at general staff meetings or in special committees? How is the staff represented? How is the administration represented? How are topics chosen?

Characteristically, it seems that when such dialogue occurs, it does not allow significant time for thorough examination and discussion of issues. Most of us are not adequately informed on important issues and good information is hard to come by. Administration always seems to be a step or two ahead of the staff in possessing knowledge of future trends; and consequently their viewpoint generally prevails.

If meetings are to be truly productive, then the medical staff needs to be well informed. Its representatives need to be able to articulate clearly and concisely the issues at hand. Furthermore, they must be capable of seeing all sides of the question, standing firm when necessary, and compromising when it is for the good of all.

Generally, there is a lack of such physician-politicians (and I use the word politician in its best sense) which needs to be addressed.

Edward J. Volpintesta, M.D.

Bethel

KEEP THE PRIMARY CARE PHYSICIAN IN THE HOSPITAL

To the Editor: Dr. Anthony Redmond's article, in the April 1992 issue of your journal, entitled *"Is the Primary Care Physician to Be Kept Out of the Hospital,"* addressed a critical issue. The most important reason for keeping the primary care physician in the hospital was not mentioned.

The healthcare of the community depends, rather rests, on the shoulders and brains of the primary care physicians. The most important element in continuing education is peer contact with a gamut of physicians on a regular basis, resulting in an interchange of ideas and information. Diagnostic acuteness, or the lack of it, will depend on the educational currentness of the physician. There are a lot of important decisions to be made in the office; the technology of medicine is increasing in complexity; more and more fractionation of medicine has led to an increase in the difficulty of choosing to whom cases should be sent; the welter of medications for a variety of purposes increases yearly. An underinformed primary care physician will refer with failures and/or unusual problems. The best informed and educated will know sooner when help is needed, or if it is needed.

Contact with in-hospital patient care, and the responsibility of the life and death needs of his patient, is the best incentive for learning and the establishment of relevance of information.

The separation of the front-line physician, which took place in portions of New York City in the 1940s and 1950s, often in medical school-influenced hospitals, produced the worst and the best care to be found domestically.

It may be easier to meet Quality Assurance goals by limiting patient care in hospitals to Board Qualified, etc. physicians, usually without reference to the need for recertification, a hot political issue; it is much easier for "leaders" if they do not have to review charts or spend time educating their "weaker" physicians—weaker may not be quantitatable by actual results—they may be "weaker" by definition, or prejudicial fiat. But the "leader" should be willing to lead, in exchange for security, and a 35-hour week, and percs, percs, percs. They don't publish much, most of them.

The estranged physician can sometimes keep up, most cannot; their relationship with the hospital and colleagues changes; they are no longer "inside"; they have lost status; they may be resentful, with some justification, after being in and working for the hospital over the years, usually without pay; they may not show up often in conferences, etc. It takes a short time to be out of date.

Younger specialists who admit, favor eviction for others—no comment. Patients are often not sent back, or may lose their relationship with their primary care physician; in subtle ways the prior care may be called to question. The next step is usually the replacement of the office/hospital physicians by "full timers" who can do no wrong, or if they do, it is not mentioned. After all, their intentions are beyond question, and they bind legally and emotionally to the parent institution.

Quality assurance will be extended to office practices some time, but by then the deterioration in care rendered by the community-based primary care physician may well be irreversible, and patients will have paid a heavy but hard-to-measure price for the greed, sloth, and envy of the "leaders," in the guise of patient care, education, government regulations, and cost.

Oh, incidentally, did you ever try to get a full timer on a weekend or night? It's either very tough, or there is a substitute; the covering doctor may be in a rather distant specialty—but that's O.K.—he has been anointed—bound to the institution emotionally and legally, and therefore above criticism. In conclusion: keep the primary care physician in the hospital.

Norman M. Canter, M.D.

Fairfield

"ATTRACTIVE EDUCATIONAL BROCHURES"

To the Editor: I hope most physicians ignore Dr. Sultan Ahamed's advice to promote animal research (President's Page, February 1993). As an attorney and adjunct professor of law, I am aware of how animal research and testing results are misused, and often do more harm than good. Consider for example the animal experiments funded by the American tobacco industry. Their sole purpose was to contradict clinical studies linking smoking to cancer. In fact, they succeeded in delaying for years the law requiring the surgeon general's warning on cigarettes, thus resulting in thousands of human deaths that might have been prevented if the warning had appeared earlier.

The so-called "attractive educational brochures" promoting animal research that Dr Ahamed wants physicians to place in their waiting rooms are undoubtedly paid for by those with vested economic interests in animal experimentation. It would be interesting to know, for example, how much funding Connecticut United for Research Excellence (CURE) receives from the U.S. Surgical Corporation.

Rather than promoting a controversial point of view that may very well alienate some patients, physicians would be better advised to provide patients with articles that might actually benefit them. Examples are: "Exercise Reduces Risk of Diabetes," *Medical World News*, July 1992, which states that weekly vigorous physical exercise may be the only practical way to reduce the risk of type II diabetes; Dr. Ornish's classic "Can Lifestyle Changes Reverse Coronary Heart Disease?," *The Lancet*, 21 July 1990, which shows that a low-fat vegetarian diet, stress reduction and moderate exercise can reverse the progression of coronary artery disease. Also appropriate would be epidemiological studies such as T. Colin Campbell's famous "China Study," *New York Times*, Tuesday, 8 May 1990, which demonstrates the relationship between a vegetarian diet and reduced rate of heart disease and colon cancer, and more recently, "Mortality Pattern of German Vegetarians After 11 Years of Follow-Up," by Jenny Chang-Claude, *Epidemiology*, September 1992, which shows a significant reduction in mortality from all causes (men, 44%, women 53%) for vegetarians as compared to the general population in Germany. Any of these would be more revelant to most patients than the material Dr. Ahamed is offering.

Finally, Dr. Ahamed could not have ended his defense of animal experimentation more inappropriately than by quoting George Bernard Shaw, a lifelong vegetarian and antivivisectionist. I feel, as Shaw did, that anyone who is capable of torturing animals in the name of science is probably also insensitive to human suffering. Dr. Ahamed should heed Shaw's words in his preface to *Doctor's Dilemma*: "Once grant the ethics of the vivisectionists and you not only sanction the experiment on the human subject, but make it the first duty of the vivisector. If a guinea pig may be sacrificed for the sake of the very little that can be learnt from it, shall not a man be sacrificed for the sake of the great deal that can be learnt from him?"

W. David Zitzkat, Esq.

Hartford

IN RESPONSE TO

"REAL REFORM OR JUST TINKERING?"

To the Editor: Your editorial in the latest (January) *Connecticut Medicine* was a balm to my troubled soul. I have long maintained that the prime course of the "health care" crisis is an insanely increased demand for our services. Why do people flock to us as they do? Just what do they think we can really do for them?

To have these issues addressed incisively and with an economy of language was refreshing. I hope people are listening.

Victor A. Altshul, M.D.
New Haven

BELLEVUE HOSPITAL EMERGENCY SERVICES 13th ANNUAL EMERGENCY MEDICINE SEMINAR

MONDAY - FRIDAY JUNE 7-11, 1993

This seminar provides registrants with a rational and systematic approach to clinical decision making in the Emergency Department. It is designed for Emergency Medicine Practitioners & for those sub-specializing in Emergency Medicine. A comprehensive syllabus complements the course.

FEE: $650
ACCREDITATION: 28 Category I Credit hours AMA/PRA

For Information write or phone:
NYU Post-Graduate Medical School
550 First Avenue,
New York, N.Y. 10016
(212) 263-5295

NYU Medical Center Post-Graduate Medical School

CM 4/93

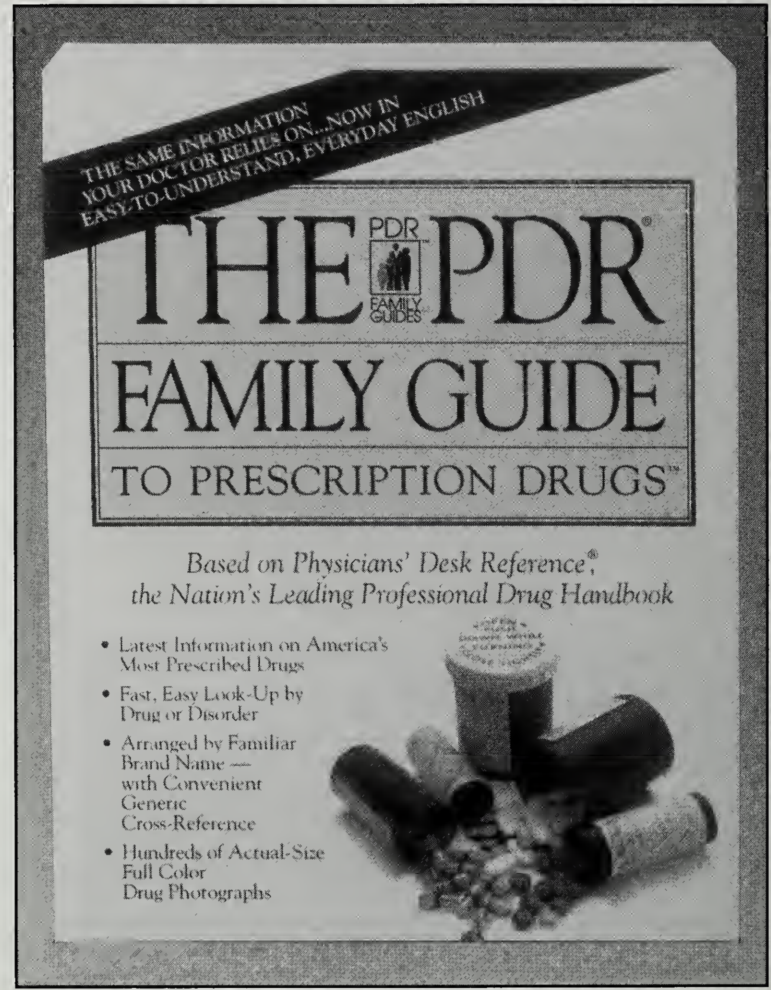
CSMS MILESTONES
1792-1992

1792	CSMS Charter and First Meeting
1793	Physician Licensure
1812	Medical Institution of Yale College
1822	Connecticut Retreat (Institute of Living)
1826	First General Hospital of Connecticut (Yale-New Haven)
1841	State Registry of Deaths and their Causes
1867	Connecticut Valley Hospital (Middletown)
1878	Connecticut State Board of Health
1893	Connecticut Medical Practice Act
1935	Connecticut Tumor Registry (World's 1st Cancer Registry)
1936	*Connecticut Medicine* (Journal of the CSMS)
1947	Connecticut Medical Service (CMS-Blue Shield)
1949	CSMS Building (160 Saint Ronan Street, New Haven)
1971	CME Accreditation of Hospitals
1974	*Connecticut Sports Medicine* Newsletter
1983	Connecticut Peer Review Organization (CPRO)
1984	Connecticut Medical Insurance Co. (CMIC)
1985	CSMS/HCMA Medicare Courtesy Card Program (Now ConnMAP)
1985	CSMS Countersuit Fund
1986	CSMS-IPA
1987	M.D. Health Plan
1988	CSMS Charitable Trust
1988	First Countersuit Pays $99,000 to CSMS Internist
1989	CSMS Physician's Guild
1990	Pictorial Directory of Connecticut Physicians
1991	CSMS Health Awareness Campaign
1992	CSMS Bicentennial

Physician's Desk Reference®
Releases New Consumer Guide
Helps People Become Medically Literate

Health-conscious Americans are demanding up-to-date information on how the medications prescribed for them impact their bodies. They want a dialogue with their doctors; they insist on participating in the decisions that affect their health. Many have purchased for their home bookshelves the same reference book that physicians have relied on since 1947, Physician's Desk Reference® The PDR is the only drug reference available that gives complete, FDA-approved guidelines for safe and effective use of prescription drugs.

The **PDR® Family Guide to Prescription Drugs**™ (ME Data, $24.95) draws on the same authoritative facts found in the PDR. It is the only drug handbook that lists drugs under their familiar brand names (ie, the way consumers commonly refer to them), then cross-reference every brand by generic name and indexes each drug by the diseases for which it is prescribed. This makes the Guide simple to use and permits fast, easy look-ups. In addition, it includes overviews of our most common diseases and disorders, and the treatments we have available today.


THE SAME INFORMATION YOUR DOCTOR RELIES ON...NOW IN EASY-TO-UNDERSTAND, EVERYDAY ENGLISH
THE PDR®
PDR FAMILY GUIDES
FAMILY GUIDE
TO PRESCRIPTION DRUGS™
Based on Physicians' Desk Reference®, the Nation's Leading Professional Drug Handbook
• Latest Information on America's Most Prescribed Drugs
• Fast, Easy Look-Up by Drug or Disorder
• Arranged by Familiar Brand Name — with Convenient Generic Cross-Reference
• Hundreds of Actual-Size Full Color Drug Photographs

PDR® FAMILY GUIDE TO PRESCRIPTION DRUGS™
ME Data — $24.95 (softcover)

Designed to enhance the doctor/patient relationship, the **PDR Family Guide to Prescription Drugs** reminds us of the questions we need to ask our doctors and pharmacists, and the basic instructions and cautions we must remember when taking prescription medications. It helps us use medications safely and effectively, alerting us to the conditions that increase the chances of a possible adverse reaction. And perhaps most important, it highlights the medical problems and conditions that might interfere with a prescription—facts about our personal health that we must make certain the doctor has in order to assure the safest, most effective treatment.

In addition to its easily-accessible, dictionary-style arrangement of drug profiles and its unique disease and disorder index, the **PDR Family Guide** includes hundreds of full-color, actual size photos of the leading brand name and generic prescription products. These can be a lifesaving aid for consumers who have mixed up their medications.

Appendices include a directory of regional poison control centers and hotline phone numbers, as well as lists of drugs that meet certain conditions, such as those that are sugar- or alcohol-free and those that may make you unusually sensitive to light.

Today, more than ever before, we are aware that we as individuals have the responsibility and the right to participate in our own health care. Modern drug therapy is a vast and complicated field—so complicated that, for many questions about medicines, the answer varies from patient to patient. The PDR Family Guide to Prescription Drugs is an aid in the cooperative effort with our doctors, alerting us to the most pertinent questions to ask and clarifying the answers we receive, thereby providing us with the tools we need to supervise our won medical care as effectively as possible.

This Month's Reading in Review

TIMOTHY B. NORBECK

- "It's hard to see patients suffer while doctors are turned into clerks as a parasitical industry (utilization review) with no rational basis for its existence attaches itself like an insatiable leech onto the carcass of medicine as we once knew it. Health care is the S&L scandal of the '90s." Philip R. Alper, M.D., a California internist

- "Managed competition" advocates point to the success experienced by the California Public Employee Retirement System (CalPERS) as evidence that such a mechanism works in containing the growth of health-care costs....CalPERS held its premium increase to 6.1% this year and projects a 1.4% hike next year....The Service Employees International Union (SEIU) contends that budgeting, not competition among the 27 plans that cover CalPERS 887,000 members, was responsible for the savings....SEIU suggests that California's fiscal crisis, which forced a restrictive spending cap on the system, is the real reason behind the premium moderation. (Author's Note: *Those who favor price controls and global budgets will most likely attempt to use this information to their advantage.*)

- Also discouraging to "managed competition" proponents is that CalPERS members did not choose low-cost plans that rate higher in satisfaction surveys....Only 5.7% elected such coverage, while 54.2% chose plans with above-average premiums....Surveys show that only 25% choose a plan primarily on the basis of costs.

- In order to cope with rising health-care costs, Quebec's Ministry of Health has proposed a user fee of $5 each time a patient uses an emergency room....It now requires a $2 service charge per prescription for those over 65 who normally get them free....A ceiling limits the fees to $100 a year.

- Ontario, which insures 10 million people or about 40% of Canada's population, has cut costs by ending coverage of electrolysis and reviewing coverage of 40 other items including psychoanalysis, vasectomies, newborn circumcision, *in vitro* fertilization, chiropractic, and podiatric services.

- According to the Workers Compensation Research Institute, Workers' Compensation costs are rising mostly because of deficiencies in the handling of claims, not because of increased claims or higher benefits....It cites growing duration of disability, more attorney involvement, and increasing lump-sum settlements...."Higher medical costs are a factor, too."

- A study released a few weeks ago by the National Center for Policy Analysis found that for each dollar the federal government spends on health care, only $.43 worth of real health services are bought....The study also noted that the government "often underestimates the actual costs of federal spending," and referred to the costs of 1972 Medicare expansions which were off by more than 233%.

- The Netherlands has stepped into the vanguard of the right-to-die movement....Its parliament approved the world's most liberal rules on euthanasia and doctor-assisted suicide....Both practices are still technically illegal, but doctors won't be charged if they notify coroners of their actions and if they follow certain guidelines (the patient must be mentally competent, must be suffering unbearable pain and request euthanasia repeatedly—and the doctor must consult a second physician before proceeding).

- *Only in America:* While intoxicated and staggering home from a party, a Chicago man felt the need to relieve himself and wandered into a shadowy area. An immigrant, he couldn't read the signs that said: "Danger," "Keep Out" and "Electric Current" Nor did a row of uneven wooden boards, laid on the ground to deter intruders, discourage him. Urinating on the Chicago Transit Authority's (CTA) Ravenswood line, where the third rail carried 600 volts of electricity, cost him his life. His family sued the CTA for negligence and was awarded $1.5 million by a jury. The appellate court tossed out the action, but the Illinois Supreme Court sustained the jury verdict. It's true that the deceased plaintiff was drunk and trespassing, and there were warning signs and wooden boards—nevertheless, the CTA was at fault. As Mike Royko said, "So now the CTA has to pay the $1.5 million and figure out a way to protect drunken men from being electrocuted when they urinate on its tracks."

TIMOTHY B. NORBECK, Executive Director, Donnecticut State Medical Society.

June 12, 1993
13th ANNUAL

ADVANCES IN GASTROENTEROLOGY

Bally's Park Place Hotel and Casino
Atlantic City, New Jersey

Sponsored by the
Presbyterian/Medical Center
Gastrointestinal Section
and the Underwood Memorial Hospital
Woodbury, New Jersey

Accreditation: Category 1 credit offered

Information: Registration Manager
SLACK Incorporated
6900 Grove Road
Thorofare, NJ 08086-9447
(609) 848-1000

CALL FOR PAPERS

Members of the *Connecticut State Medical Society* reading papers before other organizations are requested to submit their papers to the JOURNAL for consideration by the Board of Editors for publication.

Authors preparing manuscripts for submission to *Connecticut Medicine* should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office (also published in the *Connecticut Medicine*). Adherence to the instructions will prevent delays both in acceptance and in publication. Papers setup with a computer word processing program should be submitted on disk (3-1/2"/5-1/4") along with the hardcopy.

Please send them to: Robert U. Massey, M.D., *Connecticut Medicine,* 160 St. Ronan Street, New Haven, CT 06511

Unique Resource Center Described

The Connecticut Clearinghouse is the state's most comprehensive resource center for free information about alcohol and other drugs as well as related topics such as AIDS, eating disorders, and youth suicide prevention. Its holdings include books, videos, pamphlets, periodicals, and curricula, many of which address the interests of the medical community.

For example, one may borrow the video "HIV Blood Test Counseling" which provides guidelines for obtaining drug and sexual histories and for giving the patient information and counseling. Another video focuses on the importance of the patient interview in the prevention and early diagnosis of alcohol and other drug abuse.

The lending library contains books and curricula about such issues as managed care in the treatment of addictions, intervention with impaired colleagues, adolescent health care, the physician's role in the campaign against family violence, mobilizing communities for prevention projects.

The Connecticut Clearinghouse can provide pamphlets and posters for the physician's waiting room. It is also an appropriate referral for patients seeking basic alcohol and other dug knowledge information about the signs and symptoms of adolescent substance abuse, parenting skills, etc.

The Connecticut Clearinghouse, located at 334 Farmington Avenue, serves the entire state. Call 1-800-232-4424 to obtain a resource list and directories describing their holdings. The Clearinghouse is a program of Wheeler Clinic, Inc. and is funded by the Connecticut Alcohol and Drug Abuse Commission.

UNIVERSITY OF MARYLAND BALTIMORE

From the Executive Director's Office

CALL
ANNUAL MEETING OF THE HOUSE OF DELEGATES

The 1993 Annual Meeting of the House of Delegates will be held at the Ramada Inn, 275 Research Parkway, Meriden. The meeting will commence at 12:30 P.M. on Wednesday, May 12, and will continue until all business has been concluded.

11:30 Registration and Luncheon
12:30 Call to Order
Sultan Ahamed, M.D., President
Michael M. Deren, M.D., Speaker of the House
Marjorie G. Petro, M.D., Secretary

COUNCIL MINUTES

Wednesday, 24 February 1993

Attendance

Present were Drs. Ahamed, Beck, Blum, Bobruff, Brooks, Cottle, Deren, Fischbein, Franklin, Freedman, Hollister, Kamens, Keating, McDonnell, Meridy, Orphanos, Redmond, Schwartz, Sosa, Tesoro, Timmerman, Wetstone, Wolfson, Zanker. In the absence of the Chairman and the Vice Chairman, Dr. Ahamed, President, chaired the meeting.

Also present were: Mr. Norbeck, Mrs. Lindquist, Mr. Brunell, Mrs. Connelly, Mr. Sullivan, Mr. Garofalo, Mr. Thompson (FCMA), Mr. Freberg (NHCMA), Mr. Fiorentino (HCMA).

Absent were: Drs. Czarsty, Eslami, B. Fox, Hoskings, Leach, Lesnik, Parke, Petro, Sadowski, Van Nostrand, Zeppieri.

Reports on Related Organizations

CPRO: Dr. Kamens presented a slide presentation outlining a 1992 update of data which included a summary of the quality of care in Connecticut as seen through the parameters of the CPRO program.

Report on CSMS-IPA: David D. Thompson, M.D., newly elected President of CSMS-IPA, reported that he has been involved with M.D. Health Plan since its formative years. At the time of the establishment of M.D. Health Plan, there were certain goals, i.e. to preserve the way medicine was practiced by physicians of the Connecticut State Medical Society today, to maintain a reasonable fee structure so that they could pay for the services rendered, and to eliminate the hassle of private practice by cutting down on paper work, and above all to have a doctors' organization that really was operated by physicians, to see if the physicians could supply solutions to the health care problem. He believes that working together they must find a way to solve these problems, i.e. improved access, continued high quality and bringing about cost containment. A new Medical Director has been employed and is working on eliminating some pitfalls in the program and will attempt to provide better service to the providers and eliminate some of the hassle for them. They are also devising a plan that would make changes in coinsurance and create variable deductibles. It is intended that in time this plan will replace the current basic plan. They are also planning to break down to a county-wide local chapter system which it felt will give physicians more input. It will also allow locally determined fee schedules and premiums.

M.D. Health Plan: Dr. Vincent Catrini and Douglas Hayward from M.D. Health Plan joined the Council to give a report. Dr. Catrini reported that the monies that are being returned presently to physicians to catch up to the conversion system are at the level of about eighteen million dollars in the past few months. It is expected that interest will be paid on the delayed payments. There may still be some delay in processing claims that have to be corrected. The conversion in moving along at this point and M.D. Health Plan will have one of the most advanced systems in the state and will be able to take care of the problems on an ongoing basis. They welcome any input concerning problems that may be encountered. He reported that many states are looking to M.D. Health Plan as a model plan and they will be assisting other states in developing plans. It was stated that the state contract that is being sought is at the same level as previously reported and it appears that M.D. Health Plan has a very good chance of obtaining the contract, which would make M.D. Health Plan the largest HMO in the state.

Report of the President

Dr. Ahamed reported that the Board of Directors of both Hartford and New Haven Counties have expressed approval for unified county and state membership and that he and Mr. Norbeck were going to Fairfield County that evening to discuss the subject with the Fairfield County Board.

He reported that the Building Committee had met with three architectural firms to discuss preliminary plans for the building renovation. The firm of ACMAT was selected to make a feasibility study and will present a proposal at the meeting of the Building Committee on March 4 and a further report will be presented at the next Council meeting.

He brought to the attention of the Council that a Connecticut physician has named CSMS in a suit against CMIC. The question appears to be that he was refused professional liability insurance. CMIC and legal counsel is seeking to have CSMSs name removed from the suit because it was felt that we have no liability in this case.

He informed the Council that a revised video tape reviewing the Society's activities was available for review by the Council. It was decided that anyone wishing to see the tape could do so at the end of the Council meeting. It is planned to show this tape at various county meetings and if possible hospital staff meetings.

Report of the Executive Director

Mr. Norbeck reported that Kyle Ballou, who presently is out on maternity leave, has informed him that she has accepted a position with the Shawmut National Bank as Director of Public Affairs on March 22. While she was on maternity leave, several members of the staff have been picking up the slack and we will all have to continue to do this with the cooperation of Sullivan & LeShane until the end of the session. We are all sorry to see Kyle leave and wish her well. It was VOTED that a resolution be prepared and given to Kyle thanking her for her past services.

Mr. Norbeck reported on the following items:

(a) The National Center for Policy Analysis has just released a report indicating that for each dollar the federal government spends on healthcare, only 43 cents worth of real health services are bought. The Investor's Business Daily reported that the government often underestimates the actual costs of federal spending, and used the example of government forecasts for the cost of 1992 Medicare expansion which were off by 233%. The report stated that "these faulty government forecasts occurred because of a failure to realize that reducing the price people are charged would lead to a higher demand for medical services, and that higher demand would make healthcare increasingly expensive."

(b) The Oregon Medicaid waiver situation will be settled by March 17. Governor Roberts and two state legislators had pressed for a decision by mid-March so that the legislature could include another 100 million in their Medicaid budget and HHS Secretary Donna Shalala has promised to review the situation by the 17th.

(c) Brookings Institute reports that annual nursing home expenses, on average, are expected to explode from about $36,000 today to $46,000 by the year 2000, and $135,000 by 2070 based on a 5.5% annual growth rate. Long term care insurance pays less that 2% of total costs. Since 1987, only 2.4 million long term policies have been sold to nearly 30 million Americans over 65. According to the GAO, 60% of long term care insurance holders drop the policies before receiving benefits. Families USA released a report indicating that 85% of elderly Americans cannot afford long term care insurance with low premiums and high co-payments and 97% are unable to afford insurance with high premiums and low co-payments. Families USA wants Medicare expanded to include long term care. Health Insurance Association of America claims that 60% can afford long term care insurance and charges Families USA with manipulating the figures. According to confidential work papers from the President's Task Force on National Healthcare Reform, the administration is considering a new government subsidized program to provide long term care for those with chronic illness or disabilities which could cost up to 34 billion dollars over the next four years.

(d) Editorials in the New York *Times* have been pushing "managed competition" as the cure all for all healthcare. The National Journal *AARP*, Kaiser Family Foundation and others have recently criticized the *Times* for their "unbalanced" views. Business Roundtable, comprised of 200 CEOs of major corporations, endorsed managed competition along with the Governors' Association.

(e) Mr. Norbeck reported on a meeting held with Dennis May, CEO of the Connecticut Hospital Association. The CEOs of hospital and medical staff leadership have come to the conclusion, in the light of developments in other states, that it is only a matter of time before hospital and medical staff clinical and financial data will be made available to the public in Connecticut. The CHA Board plans on bringing their leadership together on April 19th, 20 to decide on strategy. Mr. Norbeck informed Mr. May that CSMS would like to be involved in this process and requested an opportunity for CSMS physicians to meet with CHA representatives before the April meeting so that CSMS could play a role in whatever strategy is adopted. It was VOTED that Drs. Ahamed, Betten, Kamens, and Meridy meet with CHA representatives prior to the April 19th meeting.

University of Maryland Baltimore

Legislative Update

Dr. Wetstone reported the deadline for introducing bills was February 23, therefore, no more bills can be raised. It was stated that there are number of bills concerning healthcare reform and they are being closely watched and that CSMS will be testifying on March 2 on these bills. A number of the issues that we are proposing are encompassed in other bills, although not critical issues, we can support these issues by supporting these bills. He reported that there are the usual bills that extend the medical practice act to non-physicians and he believed they were all dead. It was VOTED to accept the report as information.

Communications

Several suggestions were made concerning the distribution of information to the membership, i.e. computer link up with physicians' offices and forming a liaison with various staffs of hospitals. It was suggested that the Council members provide the state office with the names of physicians who could act as a liaison person and that every effort should be made to provide a name for each of the 36 hospitals in the state. In this way the information of what CSMS is doing can be brought to the attention of the medical staffs of the various hospitals.

Medical Aid for the Homeless

Leonard Vinnick, M.D., Stamford, joined the Council to discuss the problems of the homeless and described a model program that they have in Stamford. A recent survey was made on the various shelters and the vast majority have a profound need for medical help. Volunteer physicians and nurses are needed to staff the shelters, he stated, and all could choose their own hours. One solution would be for hospitals to have their house staffs provide such service, which is being done in other states. He stated that if we could approach this problem properly, it would go a long way in improving the physician's image in the state. It was VOTED to strongly support the suggestion that physicians volunteer provide medical aid for the homeless and ask the county associations to support this project. It was further VOTED to form an Ad Hoc Committee to promote this program and that the counties each provide a name to serve on this committee and that Dr. Vinnick serve as chairman.

Report of Subcommittee on Preliminary Study of Nominations

With a few amendments, it was VOTED to approve the report of the Subcommittee and to transmit the slate of nominees to the House of Delegates for election with the Council's recommendation for approval. It was further VOTED to make the following changes:

(a) Combine the Committee on Third Party Payments with the Liaison Committee with the Department of Income Maintenance.

(b) Disband the Committee on Cooperation with Medical Schools ofConnecticut.

(c) Discontinue Advisors to the Auxiliary to CSMS

(d) Change delegates to Massachusetts to President(D), President-Elect(A)

Ad Hoc Committee on Membership

Dr. Jerome Freedman stated that due to pressure of other commitments, it was necessary to submit his resignation from the subject committee. It was VOTED to accept the resignation of Dr. Freedman and expressed sincere appreciation for all his efforts in behalf of promoting membership in CSMS. It will be necessary to appoint a new chairman for this committee.

Fiscal Subcommittee

It was VOTED to accept Dr. Freedman's resignation from the subject committee and to appoint Mehdi Eslami, M.D. to the subcommittee.

Resolution Re Coverage for Psychiatric Care

Following discussion, it was VOTED to approve a resolution introduced by Theodore Zanker, M.D. which reads as follows:

Resolved, that the American Medical Association should neither endorse for sale to its members nor offer for sale to its members, either directly or through its subsidiary organizations or agencies, any health insurance program, policy or plan unless such program, policy or plan conforms to official AMA policy, including policy relating to coverage for psychiatric medical care, and be it further

Resolved, that the CSMS delegation to the AMA House of Delegates be instructed to campaign actively for the approval of this policy.

N.B.: *The foregoing is a summary of the proceedings and actions of the Council on February 24, 1993. Detailed minutes of the meeting are on file at 160 St. Ronan Street, New Haven, for perusal by any interested member of the Society.*

Testimony of the Connecticut State Medical Society

The Issue of Health Care Reform

Presented to the Public Health Committee

March 2, 1993

PRESENTED BY HOWARD WETSTONE, M.D., PRESIDENT-ELECT

My name is Howard Wetstone of Bloomfield, representing the Connecticut State Medical Society's 6,100 physician members.

I have submitted with my written testimony to the Committee a copy of the Connecticut State Medical Society's Statement on Health Care Reform which includes our Basic Health Care Plan proposal. This document outlines our position and our plan, which relies upon an employment based system.

It is the position of Connecticut's physicians that universal access to high quality medical care at reasonable cost must be assured for all citizens. This goal can be attained only with improvements in our health care delivery system and the availability of adequate financial resources. CSMS believes that modification of the existing employment based system is the best way to provide high quality medical care for all people in Connecticut.

Our proposal establishes a basic health care plan and mandates coverage for all full time employees and their families. It calls for the elimination of insurance practices which discriminate against small business and sick people by establishing community rating without preexisting conditions, portability of coverage, ERISA reform and insurance risk pools. It also puts in place methods which can achieve cost savings in the current health care delivery system such as standardized medical forms, tort reform, preventive care and inducements to expand the number of primary care physicians.

Connecticut physicians recognize the serious problems with today's health care delivery system. We deal with problems of access, cost, delivery and demand at the front lines every day. I sit before you today to again stress the Medical Society's commitment to health care reform and our willingness to participate in the reform process. What we know as physicians is how to practice medicine and we want to share our clinical and practical experience and knowledge with all those active in seeking reform.

As physicians, we feel strongly that someone who knows how to deliver care to the patient needs to be involved in the planning in any health care reform package that will work. If invited to the table the practicing physicians will bring not only our medical knowledge, but also our professional commitment to containing cost. The Connecticut State Medical Society is fully aware that there must be increased accountability within the medical profession to control cost through physician behavior. We are committed to:

- Performing professional review of excessive medical fees—if we are given the legal ability and protection to do so.
- Being subject to utilization review - if it is done in the appropriate manner under medical supervision and accountability.
- Adopting and implementing practice guidelines which will define the appropriateness of care - if such guidelines are accommodated for in tort reform initiatives.
- Utilizing outcome studies and identifying cost effective treatments..
- Working at reducing the administrative burdens and costs.

As patients, we must all take on some level of responsibility for our own health and some accountability for the health care we consume. The CSMS health plan emphasizes preventive health care, patient education and the promotion of healthy life-styles. As an organization, the CSMS supports helmet laws, excise taxes to curb alcohol and tobacco use, universal immunization, gun control, strict enforcement of speeding laws and severe penalties for drunken driving.

Ultimately, we physicians will be called upon to implement whatever reforms are decided upon. Connecticut physicians are some of the finest in the nation and we urge you not to wait to utilize this resource. We can bring our expertise to the task force to study mandated benefits called for in HB 5130 and to all other study groups and working groups focused on health care. Call upon us now and bring us to the table to help shape meaningful reform.

Testimony of the Connecticut State Medical Society The Issue of Health Care Reform

Presented to the Public Health Committee
March 2, 1993

TIMOTHY B. NORBECK

Senator Przybysz, Representative Courtney, members of the Public Health Committee, I am Tim Norbeck, Executive Director of the Connecticut State Medical Society.

As Dr. Wetstone explained in his presentation, the Connecticut State Medical Society wholeheartedly supports the concept of universal coverage.

Everyone should have health insurance and accessible care. We must also find ways to contain the growth of rising health care costs without compromising quality—and that task will be an immense challenge for all of us. All of the opinion polls show that Americans do not want to give up or compromise expensive new technology, but it is repugnant to perpetuate a system that offers miraculous medical technology to some but which fails to ensure even basic care to those without insurance coverage.

It is not possible to provide universal coverage, and state of the art quality, and lower costs. All of the experts agree: you can have any two, but not all three. Therefore, if economy is to prevail, there are difficult choices ahead. We have become an "I want it all and I want it now" society, and that attitude cannot be sustained if we are to do what we must do - provide coverage for all and come to grips with the factors fueling the engine of rising health care costs.

It was T.S. Eliot who said that "Human kind cannot bear very much reality." President Clinton is presently testing that observation with his recent challenges to all of us.

President Clinton has also already suggested that we all bear a responsibility in the rising health care costs problem. The Minnesota Health Care Commission has embraced the president's message and proclaimed that it is "stressing personal responsibility as a way of keeping health care costs down."

The Commission Chair said that "consumer responsibility was one of the early seeds of consensus" and estimates that preventible illness accounts for 70% of health spending in Minnesota and has urged Minnesotans to give up sedentary lifestyles, and to take care of their health problems early rather than waiting until they become acute. I would add to that statement that health insurance coverage for everyone must be available in order to achieve that important goal. The Commission Chair also added something somewhat controversial when he said that "there is a growing feeling in some quarters that there must be some limits on having the rest of society subsidize the health care needs that result from people who knowingly and intentionally abuse themselves."

The numbers are awesome and contribute greatly to rising health care costs. Smoking adds anywhere from $22 billion to $65 billion a year to health care costs, and tobacco use is blamed for up to 500,000 deaths a year and accounts for 32% of fatal cancers, 21% of fatal heart disease, and 88% of cases of fatal chronic lung disease. Alcohol and substance abuse add another $110 billion to our health care tab. At this point, someone might be tempted to invoke the comment of Mark Twain that "the only way to have good health is to eat what you don't want, drink what you don't like and do what you'd rather not." While he was clearly overstating the situation, it is obvious that Twain's prescription is not being filled. It is interesting to note that those residents of one of our healthiest states, Utah, enjoy the lowest overall disease rate, have the lowest incidence of cancer, and the second lowest incidence of coronary heart disease and mortality. Moreover, and probably not so coincidentally, Utah ranks lowest in consumption of tobacco and alcohol and is second highest in physical exercise.

Besides that personal responsibility, however, we must give all residents the opportunity to seek and receive appropriate care at the early onset of illness. Such important insurance coverage must encourage preventive care including early detection and screening, and immunization. I know of no better example of how important and effective a screening program can be for promoting health and containing costs than the mammogram screening program in Hawaii. Caucasian women in the Aloha State have the highest incidence rate of breast cancer in the entire United States, but, incredibly, they also have the lowest death rate from it. We are pleased to be working with the Department of Health Services' praiseworthy Coalition to Improve Breast and Cervical Cancer Screening in Connecticut.

Regardless of the health reform enacted in Connecticut and throughout the country, however, we must recognize that the growth of health care costs will not come under control until we achieve some success in addressing our nation's social problems. Poverty breeds disease, even in the most affluent of states, and we in Connecticut have a major problem in this regard. Nationally, 20 percent of our children live in poverty. Joblessness, poor housing, homelessness, ignorance, poor nutrition and violence all have such a negative impact on the quality of life and on health care costs.

We must intensify our educational efforts which also relate to health care costs. Unfortunately, our nation leads the industrial world in teen pregnancies and low-birth-weight infants. Thanks to the miracles of medical technology, preemies weighing only 20 ounces can live but can cost $2,500 a day in neonatal units and up to $1.2 million for their estimated hospital stay of 150 to 160 days. In addition, 370,000 babies, about 10% of the total number of babies born in the United States each year, have mothers who used cocaine, heroin, or amphetamines during their pregnancies. The health care costs alone in treating them are equally burdensome to society. And other major challenges await us. while searching for areas to save, eventually we will come to the complex ethical issues involving the problems unique to the first and last few years of life which reportedly account for almost 75 percent of our health care costs.

But in our zest to hold the line, we must also recognize demographic realities. As some take out their scalpels to pare down Medicare costs, let us remember that 700,000 new enrollees are added to the Medicare rolls every year.

Those 85 and over constitute the fastest growing segment of our population, increasing at a rate three times that of the population at large. Entertainer George Burns recently said that "one of the nice things about being 90 is that there's less peer pressure." That may be so, but there are many others gaining on him. We now have 37,000 people over the age of 100, doubling the number only 10 years ago. We must recognize that this happy event known as aging will also cost many more health dollars.

In closing, I would urge us all, as we attempt to preserve what is good about our health care system while fixing what needs to be repaired, to remember the old Chinese proverb, "If we don't change direction, we will get where we are headed." Almost everyone acknowledges the need to change our current direction so everyone has insurance and access to the system, but that right course also involves taking greater personal responsibility over our lives and addressing our distressing social problems. Unless we face up to these hard realities, we can never hope to contain the growth of health care costs, no matter what health care reform is enacted. Thank you.

Gala planned to celebrate Yale-New Haven Children's Hospital opening

The opening of the Children's Hospital at Yale-New Haven is scheduled for early this summer and the Friends of Yale Pediatrics, a volunteer group supporting the Children's Hospital, and the Yale-New Haven Hospital Auxiliary are preparing a Gala Ball entitled the Storybook Ball, to celebrate the opening of the building.

Joan Haversat, Meg Johnson and Mary Jo McFadden are the volunteer co-chairs for the event, scheduled for the New Haven Lawn Club on Saturday, June 19. The menu for the evening has been developed by internationally famous French chefs Jacques Pepin and Jean-Claude Szurdak. Music will be provided by Peter Duchin and his New York orchestra.

Ground was broken on the 11-story Children's Hospital at Yale-New Haven in September, 1991. The expanded pediatric service, the largest and most comprehensive between Boston and Philadelphia, will feature the state's first dedicated pediatric emergency department, pediatric operating rooms and diagnostic imaging, a 46-bed newborn special care unit, and totally revamped maternity services.

A special feature of the Storybook Ball will be the first exhibition of "Tales of Courage," an international exhibition of children's art. The exhibit will showcase the work of local area school children and children from over 200 countries. Several area art organizations are also participating in the exhibition including the New Haven Arts Council, the Creative Arts Workshop, ArtSpace and Educational Center for the Arts. Many of the art works will eventually be placed on permanent display in the Children's Hospital. For information on the Storybook Ball, contact Beth Michael at 785-3098.

UNIVERSITY OF MARYLAND
BALTIMORE

IN MEMORIAM

ALLEN, ROY, Temple University School of Medicine, 1943. Dr. Roy was a retired physician, former assistant medical examiner for West Hartford, and school physician for the American School for the Deaf. He was a member of the Hartford County Medical Association and the Connecticut State Medical Society. Dr. Allen died 15 December 1992 at the age of 76.

BRANDON, KENNETH F., University of Toronto Medical School, 1932. Dr. Brandon was a retired vice president of life underwriting at Aetna Life & Casualty Co. and a former member of the Hartford City Council. He was president of the Council of the New England State Medical Societies and former chairman and speaker for the House of Delegates in the Connecticut State Medical Society. He was a member of the Hartford County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Brandon died 30 January 1993 at the age of 83.

CANFIELD, NORTON, University of Michigan School of Medicine, 1929. Dr. Canfield was a former Yale Medical School associate professor and chief of otolaryngology, 1938-51. He was a member of the New Haven County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Canfield died 12 October 1992 at the age of 89.

DULEEP, KODENDERA S., Osmania Medical College, India 1957. Dr. Duleep was a general surgeon and a lecturer in general surgery. He was a member of the Fairfield County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Duleep died 19 December 1992 at the age of 58.

EBERS, THEODORE M., University of Nebraska School of Medicine, 1931. Dr. Ebers was a retired medical director of Connecticut Mutual Life Insurance Company, Hartford. He was a member of the Hartford County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Ebers died 3 January 1993 at the age of 86.

HAINES, HENRY L., Johns Hopkins Medical School, 1939. Dr. Haines was an otolaryngolist in New London for many years. He was a member of the New London County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Haines died 15 February 1993 at the age of 81.

IZUMI, ERNEST M., University of Texas Southwestern Medical School, Dallas, 1950. Dr. Izumi was a pathologist and medical examiner in Colebrook, Connecticut. He was also an associate professor at Yale School of Medicine. He was a member of the Litchfield County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Izumi died 25 December 1992 at the age of 75.

KELLER, LOUIS, Tufts Medical School, 1926. Dr. Keller was a specialist in otolaryngologist in Fairfield County for many years. He was a member of the Fairfield County Medical Association and the Connecticut State Medical Society. Dr. Keller died 16 November 1992 at the age of 89.

LOEWALD, HANS W., University of Rome, Italy, 1934. Dr. Loewald was a clinical professor of psychiatry at the Yale Medical School and the Yale Child Study Center. He was a member of the New Haven County Medical Association and the Connecticut State Medical Society. Dr. Loewald died 9 January 1993 at the age of 86.

LARSON, ALBERT L., Albany Medical College, 1936. He retired as vice president and chief medical director of Travelers Insurance Company. He was a member of the Hartford County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Larson died 16 December 1992 at the age of 81.

MURPHY, CHARLES A., Long Island College of Medicine, 1933. Dr. Murphy was chief of pediatrics at Stamford Hospital, Stamford, Connecticut. He was a member of the Fairfield County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Murphy died in December 1992 at the age of 86.

PIAZZA, GEORGE J., Boston University Medical School, 1932. Dr. Piazza was an allergist from 1934 until he retired in 1977. He was a member of the New Haven County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Piazza died 8 December 1992 at the age of 86.

SANTOPIETRO, OLINDO, New York Medical College, 1948. Dr. Santopietro practiced internal medicine in Waterbury for 32 years until he retired in 1984. He was a member of the New Haven County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Santopietro died 14 November 1992 at the age of 75.

WEBB, WILLIAM L., Johns Hopkins Medical School, 1955. Dr. Webb, president and psychiatrist-in-chief, made the Institute of Living Hartford one of the leading private psychiatric hospitals in the country. He was professor of psychiatry at the University of Connecticut School of Medicine and a lecturer at the Yale University School of Medicine. He was a member of the Hartford County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Webb died 14 December 1992 at the age of 62.

WEIGEL, STANLEY J., Yale University School of Medicine, 1943. Dr. Weigel practiced family medicine in Danielson for over 30 years. He was a member of the Windham County Medical Association, the Connecticut State Medical Society, and the American Medical Association. Dr. Weigel died 1 December, 1992 at the age of 78.

CSMS PHYSICIAN PLACEMENT SERVICE

The Society maintains the Physician Placement Service as a *free* service to the medical profession, hospitals, and communities in Connecticut.

***Opportunities* should be typed, double-spaced copy on letterhead and submitted to CSMS, Physician Placement Service, 160 St. Ronan Street, New Haven, CT 06511 (203) 865-0587 or fax to (203) 865-4997. These will be published as space permits and will be distributed to physicians making inquiries of such *opportunities*.**

***Physicians wishing to locate* in Connecticut may call the office requesting opportunities in their specialty. Also, candidates are invited to submit a resume to be kept on file with the Society. An announcement of a physician's availability will be published in two issues of *Connecticut Medicine* as space permits.**

Listing of physicians in the Placement Service does not in any way represent certification by the Society. Investigation of credentials and experience is the responsibility of those seeking applicants for positions.

Announcements on the Physician Placement Service page under Classified Advertising are charged at the regular Classified Advertising rate.

UNIVERSITY OF MARYLAND BALTIMORE

OPPORTUNITIES FOR PRACTICE

AMBULATORY CARE

Full-time or part-time position available for physician (Family Practice, Internist, or Emergency Medicine) in established Walk-In Center. Highly competitive salary and benefits; very pleasant working conditions; located on beautiful Connecticut shoreline in family community. Please send CV or call: Harry Ardolino, M.D., Coastline Medical Center, 9 Business Park Drive, Branford, CT 06405. Phone: (203) 481-0818.

Motivated BC/BE IM/FP/EP wanted for our unique 18 physician well established ambulatory group practice, providing quality out-patient care to the greater Hartford area from six modern urgent-care facilities. We see a mix of community and occupational clients totalling 75,000 patients each year. A close association with Hartford Hospital and University of Connecticut provides opportunities to teach residents as well as many CME activities. Excellent salary and comprehensive benefit package. Please direct inquiries to Carol Snyder, Manager of Human Resources, Immediate Medical Care Center, Silas Deane Highway, Rocky Hill, CT 06067 or call (203) 721-7393. We are an EOE.

Opportunity for partnership and a chance to practice quality medicine with your investment of time and/or capital. Walk in Medical Center in Danbury with excellent potential for rapid growth seeks interested physician willing to work to achieve success. Seasoned physician/owner seeks qualified individual to share responsibility in beautifully appointed, well equipped ambulatory care facility. Interested individuals may call in Connecticut 743-4100.

CLINICAL PHYSICIAN

The University of Connecticut Health Center/John Dempsey Hospital in Farmington seeks full or part time physician to provide primary healthcare services to UCHC students. He/She will provide clinical oversight for and evaluate a comprehensive system of "managed" care to meet health needs of students by utilizing the breadth of resources available at UCHC. He/She will participate in the education and training of medical students and housestaff. Licensure in Connecticut required; BC/BE in Internal Medicine or other appropriate primary care specialty preferred. Contact: Eileen Storey, M.D., M.P.H., Chief, Section of Occupational and Environmental Medicine, UConn Health Center, Farmington, CT 06030; (203) 679-2386. UConn Health Center is an affirmative action, equal opportunity employer.

FAMILY PRACTICE

Part time (to full time) position available in busy well established medical practice in Eastern Connecticut. Competitive salary and benefits. Pleasant working conditions with highly trained staff. Position available immediately for part time with full time in near future. Please send resume/particulars to Dr. C. Brendan Montano, 160 West Street, A-1, Cromwell, CT 06416.

Family/General Practice in Ambulatory Care Center providing acute and continuing care. Well rounded medical background and excellent patient relation skills required. Part time clinical staff position available. Competitive compensation, enjoyable work setting, pleasant ancillary staff. Send CV to David E. Wilcox, M.D., FACEP, PhysicianCare, 28 Main Street, East Hartford, CT 06118 or call for further information at (203) 569-8644.

Exercise Programs for Older Persons: Writing an Exercise Prescription

JAMES O. JUDGE, M.D.

Introduction

THE major goal of prevention from a geriatric perspective is to prolong functional independence. Physical activity and exercise may be the most effective preventive strategy health care professionals can recommend to middle-aged and older persons who wish to maintain their independence into their ninth and tenth decades. "Healthy People 2000" is the program developed by the Department of Health and Human Services and adopted by many public health groups, which has identified national goals and strategies to achieve them.[1] Just as physicians have played a major role in the reduction in stroke and heart disease mortality, we can play an important role in the next challenge of an aging society: preventing disability. An important evolution in the exercise community in the past decade has been the change in focus from "fitness, fitness, fitness!" to an approach which focuses on increased physical activity which will maintain strength, flexibility, and balance.

Physical frailty has been defined as "the state of reduced physiologic reserve associated with increased susceptibility to disability."[2] Functional tasks require a minimum (threshold) amount of flexibility, strength, balance, and aerobic capacity (fitness) for their performance. Physiologic reserve is the "safety margin" between maximal performance and the minimum required for function. Older people approach thresholds of physical capability because increasing age, sedentary life style, or the burden of chronic disease reduce their physiologic reserve.[2] Functional activities may require near-maximal or supra-maximal performance, and will be avoided. Certain functional activities require specific levels of strength, balance, and flexibility to perform. For example, getting out of a bath tub independently requires balance, flexibility, and strength. Other activities, such as shopping or walking, require stamina, or the ability to perform an activity over a prolonged period, and are dependent on endurance (aerobic capacity, or Vo_2 max).

Role of Physical Activity in Prevention

Almost half of the older population engages in no regular physical activity.[3] In the past 10 years there has been a trend toward increase in the proportion of older persons engaging in regular physical activity.[4] In prospective studies, lack of usual physical activity or reduced fitness are independent risk factors for all-cause and cardiovascular mortality.[5-7] In one study, the type of activity was not recorded, only the estimated caloric output from daily activities, where vigorous activities were not common. The greatest risk reduction was between the groups with the lowest fitness (activity) and the groups of average fitness (activity).[5,6] The differences between average and the most fit were substantially smaller, consistent with the law of diminishing returns. Two large prospective studies in middle-aged to older males found that physical activity prevented the development of diabetes mellitus.[8,9] The overall risk of diabetes was 0.61 in men who exercised at least once per week.[9] The greatest benefits (risk reduction) were found in overweight men (body mass index = weight [Kg]/height^2 [m^2] $> 25^8$ or 26.4^9) at high risk for developing diabetes, where the risks were reduced to 0.43 and 0.39, respectively, of the rate in inactive men. These results are encouraging. Exercise, or regular physical activity which was not effective in achieving normal weight, reduced the incidence of diabetes by about 60%. Physicians who have not been successful in inducing a patient to lose weight should encourage the overweight patient to be physically active. The patient does not need to lose weight to have a positive health benefit.

JAMES O. JUDGE, M.D., Assistant Professor of Medicine, University of Connecticut School of Medicine, Farmington.

The public health implications from these large-scale, prospective studies are clear. People can derive substantial health benefits from moderate levels of physical activity. That is, the health benefits accrue from levels of physical activity which do not result in athletic levels of fitness. Whereas in the past, exercise prescriptions have emphasized the need to work at an intensity that will cause an endurance training effect, the epidemiologic evidence supports a prescription to increase physical activity at a intensity that may be below the level to increase fitness substantially.

Of greatest importance to older persons, physical activity prevents or delays functional loss.[10,11] After correcting for premorbid conditions, lack of usual physical activity (never walking a mile or no regular exercise) increased the risk of future functional loss by 50%.[10] Declines in functional status are also closely related to chronic disease burden.[11] Physical activity may delay or reduce disability by lowering the prevalence of chronic diseases. Additional benefits include maintaining endurance and body composition (muscle mass, percent body fat, and bone mineral density).[12-14]

Although physical activity is effective in preventing disease and disability in prospective studies, the effectiveness of interventions which increase activity in older sedentary persons is only now being tested. A recent study has reviewed the effects of exercise interventions in older adults.[15] Targeted exercise interventions can improve muscle strength, body composition, fitness, and measures of balance. Interventions performed by investigators at the University of Connecticut School of Medicine have demonstrated improvements in balance function, muscle strength, gait, and bone mineral density in older subjects.[13,16,17]

Components of Function

Strength.—Muscle mass declines between 0.5% and 1.0% annually in men and women over 60 years.[18,19] Cross-sectional studies have demonstrated a 20-40% decrease in strength from the third to the eighth decades.[20-22] These declines in strength may be greater than the loss of muscle mass.[23,24] Physical activity can preserve the relationship between muscle mass and muscle force. Older weight lifters and runners generate greater force per unit of muscle cross sectional area than sedentary controls.[25]

Strength decrements are associated with declines in performance measures. Diminished strength in the lower extremities was found in residents of a long-term care facility with a history of two or more falls in the previous year compared to those who had not reported a fall.[26] "Fallers" had severe loss of ankle strength, particularly in dorsiflexion. Leg weakness has also been associated with impaired gait function (slow velocity and small steps).[17,27]

Table 1.—Healthy People 2000 Goals

1. Decrease the proportion of people over 65 years who engage in no leisure time physical activity from 43% to 22%.
2. Reduce the proportion of people over age 65 who have difficulty performing two or more personal care activities (ADL) from 11.1% to 9%.
3. Increase the proportion of physicians who are knowledgeable in prescribing an activity prescription from 30% to 50%. The exercise prescription should specify the intensity, frequency, duration, and type of activity.

Effects of strength (resistance) training.—Short-term supervised resistance training can increase strength substantially, and can be tolerated by older persons. A 12-week resistance training program of knee flexion and extension in older men (60-72 years) resulted in a 10-18.5% increase in isokinetic knee moments and a 107-227% increase in 1 RM (one repetition maximum), and an 11% increase in mid-thigh muscle area.[28] In older women (64-86 years), 12 weeks of resistance training improved lower extremity strength 28-115% of the initial 1 RM.[29] Neither study demonstrated a good correlation between increases in strength and muscle cross-sectional area. Resistance training in nursing home subjects (mean age= 90 years) improved knee extensor 1 RM by 174±31% and increased the cross-sectional area of thigh muscle by 9%.[30] Comparable results have been reported in upper extremity resistance training.[31]

Gains are greatest when the speed, range of motion, and type of force generated during training most closely resembles the procedures used during assessment.[32] This explains the discrepancy between the large improvements in maximal weight lifted (1 RM) which occur as a result of weight training as compared to the modest increases in isokinetic strength and muscle mass. The specificity of resistance training suggests that to improve functional status, resistance training should approximate functional tasks in joint speed, range of motion, and type of force generated during resistance training.

Flexibility

Restrictions in joint range of motion are common in older persons and are usually due usually to tight muscles rather than to fixed joint contractures. Tightness of the calf muscles restrict ankle dorsiflexion, tightness of hip flexors are common in persons with kyphosis, tightness in hip adductors and hip abductors are also frequent. Flexibility programs are effective in increasing the range of motion in the shoulder[33] and the spine.[34]

Table 2.—Exercise Prescription Principles

1. The exercise program should fit the goals of the patient.
2. The program must be adapted for the existing limitations of a person.
3. The pace of the program should be slowly progressive.
4. Introduction of new activities should follow the mastery of a given activity. Otherwise, musculoskeletal injury is likely.

Functional outcomes of resistance/flexibility programs.—A 12-week program of lower extremity resistance, flexibility, and balance exercises in life-care community residents (mean age 82) increased isokinetic knee extension 25%, and usual gait velocity 8% from 1.04±.07 to 1.12±.06 m/s^{-1}. Training strength and balance achieved a clinically significant improvement in gait velocity, despite the absence of a gait training component.[17] We have also found that combined resistance/endurance/ flexibility/ balance program improved single stance balance measures in women aged 63-77.[16]

Endurance

Fitness, usually expressed as $\mathrm{Vo_2}$ max, declines with aging in sedentary people and athletes, but older athletes have fitness levels comparable to sedentary persons 20-30 years younger.[35] Fitness declines in parallel with changes in body composition associated with sedentary aging. More than half the loss in fitness with age is explained by loss of muscle mass.[36]

The response to endurance training, when measured as percentage improvement in fitness ($\mathrm{Vo_2}$ max) is similar in young and old persons, when the relative intensity of training was similar.[37] While walking was generally well tolerated,[18] high rates of musculoskeletal injury occur when running or jogging is introduced.[33]

Interactions of endurance and strength.—The association of muscle mass and fitness noted above provides an opportunity for training in either domain (strength or fitness) to improve physiologic reserve in the other. For example, while resistance training does not improve endurance function in young subjects,[39] 10 weeks of heavy resistance training in older men increased aerobic power of the leg (oxygen consumption on a bicycle ergometer).[40] Two recent studies have found that endurance programs improve strength, maximal work, and muscle cross-sectional area. A long-term program of aerobic training and low-resistance exercise in older women (mean age 72) improved $\mathrm{Vo_2}$ max 16%, isokinetic strength 6.5%, and muscle cross-sectional area.[41] Endurance training in older men improved $\mathrm{Vo_2}$ max 38%, and improved maximal isokinetic work on a cycle ergometer by 12%.[42] Prolonged endurance training in sedentary subjects aged 60-75 years reduced body fat and maintained or increased muscle mass.[43,44] Combined endurance and light resistance training improved fitness and strength measures in a primary prevention trial in older men.[29]

Exercise Prescription

The physician prescribing increased physical activity program for his patients should develop a protocol that will increase the likelihood of maintenance of increased physical activity, with the avoidance of injury. Compliance with the prescription will require understanding by the physician of the short- and long-term goals of the patient; the goals of the program should be patient-centered. The patient is exercising with specific goals which have meaning and worth for him or her. The exercise principles listed in Table 2 can serve as a checklist as one develops the exercise prescription. Developing an exercise prescription form with a check-list of items listed in Table 3 will demonstrate the importance the physician attaches to increased physical activity.

Flexibility.—Flexibility exercises are an important component of an exercise program, primarily to reduce the risk of muscle-tendinous injuries during other components of an exercise program.

Flexibility exercises usually include static stretching (30 sec) of multiple muscle groups, particularly the hamstrings, gastrocnemius-soleus, hip flexors, and hip abductors/adductors. Upper extremity and shoulder girdle flexibility are equally important to maintaining independence in activities of daily living (ADL), especially dressing, and grooming (washing hair).

Strength Training.—Resistance training is designed to increase the force that a muscle group can generate, and is most important in persons who are beginning to have difficulty in functional activities that require strength and balance. The primary preventive role of resistance training has not been determined. Resistance training includes both isolated muscle group contractions as well as multiple-joint movements similar to functional activities like rising from a crouch, step ups, or lifting objects.

To increase muscle force (strength) substantially, exercising the muscle to a state of fatigue appears to be necessary.[32] The intensity of resistance training varies between studies, but most studies that have found substantial strength improvements trained muscles at greater

Table 3.—℞: Components, Frequency, Intensity, Duration

A. Flexibility	
1. Lower extremity	
Hamstrings	
Gastrocnemius/soleus	
Hip adductors, hip flexors	
2. Spine/ standing posture	
Rotation	
Extension	
3. Upper extremity -Shoulder girdle	
4. Functional training/ balance	
a. Transfers from sitting to standing, up from a crouch.	
b. Pick up objects from the floor.	
c. Get down on floor mat, roll prone-supine, and get up from the mat.	
d. Forward-backward, side-to-side leaning, turning to the side.	
B. Strength	
1. Frequency:	2-3 times per week.
2. Duration:	About 30 minutes, or about fivemuscle groups per session.
3. Intensity:	Dependent on functional goal. Moderate intensity (about 60-70% of 1 RM-for maintenance of strength). Higher intensity (75-80%) for strength gains. Do not exceed 80%.
4. Training Mode:	Isolated muscle exercises *v.* multiple joint exercises in movements similar to difficult functional tasks. Sandbag resistance or resistance machines.
C. Endurance	
1. Frequency:	≥ 4 times per week.
2. Duration:	30-60 minutes eventual goal. 10 minutes in frail persons initially.
3. Intensity:	Moderate (use of Borg scale of rating of perceived exertion) recommend RPE of 11-13 (on 6-20 scale), "somewhat light" to "somewhat hard."

than 70% of the maximum force the muscle can generate, and the muscle is exercised until fatigue or failure to complete the movement occurs. Each group of contractions to fatigue is termed a "set." The number of "sets" performed during a training session varies. Three sets are frequently performed, but protocols vary from two to five or more sets during a training session.

Sandbag resistance can be effective resistance stimulus for most individuals in specific exercises (for example hip extension, hip abduction, knee flexion). Persons with significant weakness in the quadriceps can benefit from sandbag resistance training, but stronger persons will require resistance machines to achieve increases in muscle force at the quadriceps. A recent review of specific resistance training techniques in older persons may be helpful for specific training issues.[45] Resistance training is now part of most supervised cardiac rehabilitation programs, as the hemodynamic responses to resistance training are comparable to or lower than endurance exercise.[46] For untrained older subjects, working with an exercise specialist is probably indicated at the initiation of training.

Endurance or prolonged physical activity.—The benefits from increased, prolonged physical activity include improved endurance (duration of an activity without fatigue), maintenance of body composition, and probably stimulation of balance control in walking programs. If the increased fitness reduces the perceived difficulty of performing an activity, that activity is more likely to be undertaken.

Activities that are likely to increase endurance include: walking, swimming, golf (not using a cart), bicycles, and folk and ballroom dancing. Rowing machines and cross-country ski machines are also used by some older persons. Jogging is not recommended in individuals who have not been runners in recent years.

The general recommendation is to begin the activity at a moderate pace; defined as a pace a person can continue for about 10 minutes without fatigue. If a person is walking at a slow pace and is fatigued earlier, the initial goal is, nevertheless, to continue the regular performance of the activity, slowly increasing the duration as tolerated. In most cases the person will be able to perform an activity for 10 minutes after several weeks. Continued fatigue before 10 minutes would raise concerns about underlying cardiac, pulmonary, or musculoskeletal limitations which need investigation. Adaptations of the exercise program for musculoskeletal limitations are frequently required. Persons with knee or hip arthritis will benefit from an exercise program,[47] but special care should be given (see section Identifying musculosketal problems).

A method for prescribing exercise intensity has evolved only recently. Pulse rate prescriptions, using predictions based on nomograms of maximum heart rate, are being deemphasized by some exercise leaders. Pulse rate nomograms have significant predictive errors because of the physiologic variability of maximum heart rate in older persons. Also, many older people have difficulty measuring and calculating their pulse rate. Some exercise leaders now use the Borg "Rating of Perceived Exertion Scale" (RPE) as a method for exercisers to monitor the exercise intensity.[48] This rating scale reflects the subjective assessment of exertion, and is a reliable predictor of the heart rate response during treadmill testing. Rather than stopping to take his pulse, the exerciser focuses on the perceived difficulty of the activity. The rating varies from "very, very light–6 to "very, very hard"–20. Exercisers can be taught to keep the exercise in the "fairly light" to "somewhat hard" range, which corresponds to an exercise intensity of about 55-70% of maximal oxygen uptake.[48]

Preexercise Evaluation

Identifying musculoskeletal problems.—Most patients will gain a great deal simply by buying a comfortable pair of walking shoes, starting a walking program at a comfortable pace, and over a number of weeks, increasing the duration of the walk, keeping the intensity (perceived difficulty) the same. If a training effect occurs, the person will walk faster and for a longer period, but the perceived exertion will remain the same. For many patients, addressing reduced flexibility, weakness, and joint restriction or joint pain is necessary. The screening musculoskeletal examination should identify restrictions in range of motion (ROM), weakness, and painful activities. Identified problems are usually best addressed by working with a physical therapist or an exercise leader with experience in working with older persons. Often several sessions with a therapist to develop an individualized stretching program will permit persons with limitations to participate fully in a group-based or individually-paced exercise program.

Cardiac disease and cardiac risk factors.—Regular exercise is recommended for most patients with stable cardiac disease. There is uncertainty regarding preexercise testing for older persons. In asymptomatic persons with two or more risk factors, a stress test is recommended by most authorities before starting a vigorous exercise program.[49] However, the present recommendation for sedentary persons is not to start a vigorous exercise program, but to increase physical activity in nonvigorous pursuits. It is important to differentiate between increases in usual physical activity and performing strenuous exercise. Activities which do not raise the double product (pulse x systolic blood pressure) substantially are less likely to induce cardiac ischemia due to fixed coronary artery lesions than vigorous activities. There is no evidence to support the need for a screening exercise stress test in asymptomatic persons before starting a moderate intensity walking program.

Exercise Setting

The setting is less important than the frequency of the exercise.[50] In a large long-term exercise study, subjects exercising on their own had similar compliance and fitness improvements to those in group-based exercise programs. Many older persons enjoy the socializing which is part of a group exercise program, and socialization may help long-term maintenance of increased physical activity.

In Connecticut all shopping malls permit walking before the stores open in the morning. Many malls have more than 100 regular walkers daily. The advantages of a climate-controlled setting with a fairly safe (obstacle free) walking surface are positive factors which may contribute to the popularity of the malls for walking. Some retirement communities without exercise facilities provide transportation to a mall for this purpose.

Conclusion

The importance of regular physical activity in maintaining independence is well-supported. Increasing the number of older persons who are physically active will undoubtedly be helped by the advice and support of their physicians. Physicians can accelerate the trend toward increasing in usual physical activity, especially in their patients who are least active and who will likely have the greatest health benefits from increasing activity. Physicians can also play an important role by supporting efforts on a town and regional basis to increase the availability of walking or bicycling trails which will make increased physical activity more accessible and attractive to sedentary persons.

REFERENCES

1. Healthy People 2000. U.S. Dept. of Health and Human Services, Public Health Service 1991; Washington D.C., U.S. Government Printing Office, 1991.
2. Buchner DM, Wagner EH: Preventing frail health. *Clin Geriatr Med* 1992; 8:1-17.
3. Wagner EH, LaCroix AZ, Buchner DM, et al: Effects of physical activity on health status in older adults l: Observational studies. *Annu Rev Publ Health* 1992; 13:451-68.
4. Stephans T: Secular trends in physical activity: Fitness boom or bust: *Res Quart Exerc Sport* 1987; 58:94-108.
5. Paffenbarger RS, Hyde RT, Wing AL, et al: Physical activity, all-cause mortality, and longevity of college alumni. *N Engl J Med* 1986; 314:605-613.
6. Blair SN, Kohl HW, Paffenbarger RS, et al: Physical fitness and all-cause mortality. *JAMA* 1989; 262:2395-401.
7. Rakowski W, Mor V: The association of physical activity with mortality among older adults in the longitudinal study of aging (1984-1988). *J Gerontology* 1992; 47:M122-9.
8. Helmrich SP, Ragland DR, Leung RW, et al: Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N Engl J Med* 1991; 325:147-52.
9. Manson JE, Nathan DM, Krolweski AS, et al: A prospective study of exercise and incidence of diabetes among US male physicians. *JAMA* 1992; 268:63-7.
10. Mor V, Murphy J, Masterson-Allen S, et al: Risk of functional decline among well elders. *J Clin Epidemiol* 1989; 42:895-904.
11. Jette AM, Branch LG, Berlin J: Musculoskeletal impairments and physical disablement among the aged. *J Gerontol Med Sci* 1990; 45:M203-8.
12. Kohrt WM, Obert KA, Holloszy JO: Exercise training improves fat distribution patterns in 60-70 year old men and women. *J Gerontology* 1992; 47:M99-M105.
13. Dalsky GP, Stocke KS, Ehsani AA, et al: Weight-bearing exercise training and lumbar bone mineral content in postmenopausal women. *Ann Internal Med* 1988; 108:824-8.
14. Morey M, Cowper PA, Feussner JR, et al: Two-year trends in physical performance following physical exercise among community-dwelling older veterans. *J Am Geriat Soc* GS31991; 39:549-554.
15. Buchner DM, Beresford SAA, Larson EB, et al: Effects of physical activity on health status in older adults II: Intervention Studies. *Ann Rev Publ Health* 1992; 13:469-88.
16. Judge JO, Lindsey C, Underwood M, et al: Balance improvements in older women: Effects of exercise training. *Phys Ther* 1993 (in press).
17. Judge JO, Underwood M, Gennosa T: Exercise improves gait velocity in older persons. *Arch Phys Med Rehabil* 1993 (in press).
18. Aloia JF, McGowan DM, Vaswani AN, et al. Relationship of menopause to skeletal and muscle mass. Am J Clin Nutr 1991; 53:1378-83.
19. Flynn MA, Nolph GB, Baker AS, et al: Total body potassium in aging humans: A longitudinal study. *Am J Clin Nutr* 1989; 50: 713-7.
20. Larsson L: Aging in mammalian skeletal muscles. In *The Aaina Motor System* (Eds.) Mortimer JA, Pirazzolo FJ, Maletta GJ, pp. 60-96, NY: Praeger, 1982.
21. Murray PM, Duthie EH, Gambert SR, et al: Age-related changes in knee muscle strength in normal women. *J Gerontology* 1985; 40:275-280.
22. Stalberg E, Borges O, Ericsson M, et al: The quadriceps femoris muscle in 20 70-year-old subjects: Relationship between knee extension torque, electrophysiologic parameters, and muscle fiber characteristics. *Muscle and Nerve* 1989; 12:382-9.
23. Frontera WR, Hughes VA, Lutz KJ, et al: A cross-sectional study of muscle strength and mass in 45- to 78- yr-old man and women. *J Appl Physiol.* 1991; 71(2):644-50.
24. Kallman DA, Plato CC, Tobin JD: The role of muscle loss in the age related decline of grip strength: Cross-sectional and longitudinal perspectives. *J Gerontol* 1990; 45,3:M82.
25. Klitgaard H, Mantoni M, Schiaffino S, et al: Function, morphology and protein expression of ageing skeletal muscle: A cross-sectional study of elderly men with different training backgrounds. *Acta Physiol Scand* 1990; 140:41-54.
26. Whipple RH, Wolfson LI, Amerman PM: The relationship of knee and ankle weakness to falls in nursing home residents: An isokinetic study, 1987. I 35:13-20.
27. Judge JO, Smyers D, Wolfson L: Muscle strength predicts gait measures in older adults (abstract). *J Am Geriatr Soc* 1992; 40(10):SA27(p 30).
28. Frontera WR, Meredith CN, O'Reilly KP, et al: Strength conditioning in older men: Skeletal muscle hypertrophy and improved function. *J Appl Physiol* 1988;64:1038-44.
29. Charette SL, McEvoy L, Pyka G, et al: Muscle hypertrophy response to resistance training in older women. *J Appl Physiol.* 1991; 70:1912-16.
30. Fiatarone MA, Marks EC, Ryan ND, et al: High intensity strength training in nonagenarians: Effects on skeletal muscle. *JAMA* 1990; 263:3029-34.
31. Brown AB, McCartney N, Sale DG: Positive adaptations to weight-lifting training in the elderly. *J Appl Physiol* 1990; 69:1725-33.
32. Fleck SJ, Kraemer WJ: *Designing Resistance Training Programs.* Champaign, IL, Human Kinetics Books, 1987.
33. Rabb DM: Light resistance and stretching exercises in elderly women: Effect of flexibility. *Arch Phys Med Rehabil* 1988; 2:688-72.
34. Rider RA, Daly J: Effects of flexibility training on enhancing spinal mobility in older women. *J Sports Med Phys Fitness* 1991; 31:214-7.
35. Pollock ML, Foster C, Knapp D, et al: Effect of age and training on aerobic capacity and body composition of master athletes. *J Appl Physiol* 1987; 62(2):725-31.
36. Fleg JL, Lakatta EG: Role of muscle loss in the age-associated reduction in $\text{Vo}_{2\ \max}$. *J Appl Physiol* 1988; 65:1147-51.
37. Kohrt WM, Malley MT, Coggan AR, et al: Effects of gender, age, and fitness level on response of $\text{Vo}_{2\ \max}$ to training in 60-71 year olds. *J Appl Physiol* 1991; 71:2004-11.
38. Pollock ML, Carroll JF, Graves JE, et al: Injuries and adherence to walk/job and resistance training programs in the elderly. *Med Sci Sports Exerc* 1991; 32:1194-200.
39. Dudley GA, Fleck SJ: Strength and endurance training: Are they

mutually exclusive? *Sports Med* 1987; 4:79-85.

40. Frontera WR, Meredith CN, O'Reilly, et al: Strength training and determinants of $Vo_{2\ max}$ in older men. *J Appl Physiol* 1990; 68: 329-33.
41. Cress ME, Thomas DP, Johnson J, et al: Effects of training on $Vo_{2\ max}$, thigh strength, and muscle morphology in septuagenarian women. *Med Sci Sports Exerc* 1991; 23,6:752-8.
42. Jones NL, McCartney N: Influence of muscle power on aerobic performance and the effects of training. *Acta Med Scand* 1986; suppl 711:115-22.
43. Kohrt WM, Obert KA, Holloszy JO: Exercise training improves fat distribution patterns in 60-70 year old men and women. *J Gerontology* 1992; 47:M99-M105.
44. Pramenko JM, Judge JO: Relationships of lean muscle mass and strength following prolonged resistance training in older women. *J Am Geriatr Soc* 1991; 39:A54.
45. Judge JO: Designing resistance training programs. *Topics in Geriatric Rehabilitation* 1993; 8:38-50.
46. Verrill D, Shoup E, McElveen G, et al: Resistive exercise training in cardiac patients. *Sports Medicine* 1992; 13:171-93.
47. Kovar PA, Allegrante JP, MacKenzie CR, et al: Supervised fitness walking in patients with osteoarthritis of the knee. *Ann Int Med* 1992; 116:529-34.
48. Borg GA: Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982; 14:377-81.
49. Schlant RC, Blomqvist CG, Brandenburg RO, et al: Guidelines for exercise testing. A report of the joint American College of Cardiology/American Heart Association Task Force on Assessment of Cardiovascular Procedures. *Circulation* 1986; 74:653A-67A.
50. King A, Haskell WL, Baylor CB, Kraemer HC, DeBusk RF: Group- *vs* home-based exercise training in healthy older men and women. *JAMA* 1991; 266:1535-42.

You'll love working with our locum tenens physicians and allied health care professionals.

WE GUARANTEE IT.

CompHealth has thoroughly credentialed physicians and allied health care providers from more than 40 fields of specialization available to provide locum tenens, or temporary, staffing assistance when and where you need it.

Plus, we have the standards and experience to guarantee your satisfaction each time we place a member of our medical staff in your practice or facility. It's the closest thing you'll find to a risk-free way to cover for absent staff members, "try out" a potential new recruit, or take care of your patients while you search for a new full-time associate.

Call us today to arrange for quality locum tenens coverage, or to discuss your permanent recruiting needs.

CompHealth

COMPREHENSIVE HEALTH CARE STAFFING

1-800-453-3030

Salt Lake City ■ Atlanta ■ Grand Rapids, Mich.

UNIVERSITY OF MARYLAND AT BALTIMORE

Because One Size Doesn't Fit All...

Eli Lilly and Company can suit all your needs with the most complete line of human insulins available.

Featuring Humulin 70/30* and our latest addition to the premixed line, Humulin 50/50†—especially useful in situations in which a greater insulin response is desirable for greater glycemic control.

Humulin®
human insulin
(recombinant DNA origin)

Tailor-made options in insulin therapy

WARNING: Any change of insulin should be made cautiously and only under medical supervision.

*Humulin® 70/30 (70% human insulin isophane suspension, 30% human insulin injection [recombinant DNA origin]).

†Humulin® 50/50 (50% human insulin isophane suspension, 50% human insulin injection [recombinant DNA origin]).

Lilly

Global Excellence in Diabetes Care
Eli Lilly and Company
Indianapolis, Indiana
46285

HI-7918-B-349310 © 1993, ELI LILLY AND COMPANY

Driving Safety in Elderly Individuals

RICHARD A. MAROTTOLI, M.D.

ABSTRACT—Driving safety in elderly individuals is becoming an increasingly important issue in geriatrics and in medical practice. The number of elderly drivers is increasing as the population ages, and especially as current generations of female drivers age.[1] Concern is raised about their safe operation of motor vehicles because of the increasing likelihood with advancing age of developing conditions that may adversely affect the visual, cognitive, and motor abilities integral to driving. But this issue is not only a medical one, since there are social and political components as well. This discussion will describe the background of this issue, focus on the changes that may occur with aging and their potential relationship to driving ability, and, finally, will outline an approach that physicians may employ in their practice.

Introduction

THE driving safety of elderly individuals has come to the public's attention in recent months following a *New York Times* front page article describing two incidents in which older drivers lost control of their vehicles and crashed into crowds of people.[2] Since driving is a task with many components, it is conceivable that performance of some of these component tasks may deteriorate with aging due to impairments in a variety of organ systems. It is not fair to condemn all older drivers since age alone is not the determining factor. Certain older individuals, however, have particular combinations of impairments that affect driving ability.

Surviving members of the Yale Health and Aging Project, a representative cohort of community-living elders in New Haven initiated in 1982, had a driving questionnaire administered in 1989. Of the 1,331 survivors, 34% were still driving, 23% had stopped driving, and 43% had never driven. Women comprised 88% of the group that had never driven.[3] The absolute number of crashes involving older drivers is low. However, since older individuals drive fewer miles, when adjusted for mileage, the crash rates for older individuals increase. Mileage-adjusted crash rates are highest for the youngest drivers, lowest for the middle-aged drivers, and begin increasing again after age 60.[4,5]

Of greater concern than the high mileage-adjusted crash rates is the increased likelihood of injuries and death from these crashes in older individuals compared to younger individuals in crashes of similar magnitude.[4,6] Indeed, "accidents" (which include falls as well as crashes) are among the leading causes of death in older individuals.[7] The Northeast Ohio Trauma Study found that individuals age 75 years and older had twice the hospital admission rate and case fatality rate of individuals less than 65 years old following a crash.[8,9] This is most likely due to the decreased reserve capacity of organ systems with aging, thereby lessening an older individual's ability to withstand similar amounts of trauma and increasing the likelihood of death from the trauma or from complications. Because of this increased risk of injury and death, efforts have been made to identify the factors that contribute to both driving ability and driving safety among older drivers. Most of this effort has been directed at the driver, although vehicular and environmental factors also play a role. Visual, cognitive, and physical function are the primary areas of concern when assessing the factors that affect driving ability.

Numerous medical conditions can affect driving ability either acutely or progressively over time. Any condition (typically cardiac, pulmonary, or cerebrovascular) that reduces blood flow or oxygen supply to the brain can affect driving ability. Fixed or progressive neurological disorders that affect cognitive and/or motor ability (such as Parkinson's disease, stroke, or dementia of any etiol-

---

RICHARD A. MAROTTOLI, M.D., M.P.H., Section of Geriatrics, Department of Internal Medicine, Yale University School of Medicine, New Haven; West Haven VAMC, West Haven.

ogy) may also impair driving. Additionally, a number of transient disorders (hypoglycemia, transient ischemic attacks, seizures, or syncope) may have an acute effect on driving ability.[10-12]

Various drugs can have an adverse effect on an individual's ability to operate a motor vehicle. Alcohol is the number one contributor to fatal accidents in North America.[13] Although alcohol use plays a prominent role in motor vehicle crashes involving younger drivers, older drivers are affected as well. Of importance is the greater effect on cognitive and motor performance from the same amount of alcohol in older individuals.[14] While illicit drug use is infrequently encountered among older individuals, numerous prescription and nonprescription medications with psychoactive properties can affect driving ability. Among these are antihistamines, antipsychotics, antidepressants, anxiolytics, sedative-hypnotics, and analgesics.[15]

Visual, cognitive, and physical function are the primary areas of concern because they are integral to driving ability; by impairing these functions many medical conditions and medications exert their adverse effect on driving ability. Particular elements of visual function are adversely affected by age-related ocular disorders such as cataracts, glaucoma, macular degeneration, and diabetic retinopathy.[16] Five to 10% of drivers ages 60 to 65 have corrected static acuity less than 20/40.[17] Johnson and Keltner showed that the incidence of visual field loss was nearly four times higher after age 65 than in those less than 60, and more than half of these individuals were unaware of their visual field deficits. Visual field loss in both eyes resulted in twice the accident and conviction rates of unaffected drivers.[18] Dynamic visual acuity, the ability clearly to identify objects moving at an angle, has also been shown to be an important element of driving ability.[19] Other visual problems that older individuals face include progressively increasing difficulty with glare from overhead road lights or oncoming vehicles and markedly impaired vision in low illumination situations.[17] A study by Sivak et al showed that individuals age 60 and older were only able to read road signs at night at two thirds the distance of younger individuals.[20]

The physician's role is to encourage older patients to have periodic evaluation of their vision (at least every two years in the absence of problems) and to correct or treat any underlying problems when possible. The generally accepted minimal vision standards for driving are static acuity of at least 20/40 (corrected) and binocular visual fields of 140 degrees or greater.[21] Many states require vision testing with license renewal, and some also require written and/or driving tests. In July 1993 Connecticut is scheduled to begin requiring vision testing of all drivers at every other license renewal (every eight years).

Hearing is another important sensory element to driving ability, although hearing-impaired individuals often make more use of visual cues and are more alert to compensate for the deficit.[11] However, older individuals are at risk for combined hearing and vision impairment.

The effects of normal aging on cognitive ability typically have minimal effect on driving capacity. However, numerous studies have shown the detrimental effects of dementia on driving ability.[22-24] Memory deficits, impaired judgment, and poor safety awareness are all likely to adversely affect driving. The latter factor, poor safety awareness, is of particular concern since demented individuals are typically less aware of their deficits and may get into risky driving situations. In contrast, individuals with visual or other physical impairments are often aware of these deficits and make adjustments in their driving patterns to compensate (for instance, driving less at night or during rush hour).

The assessment of cognitive function may be done in several ways, although the quickest way is to use a brief, standardized instrument such as the Folstein Mini Mental State Examination (MMSE).[25] The MMSE assesses orientation, short-term memory, attention, language, and constructional ability. Unfortunately, what is not clear is an appropriate cut-off point beyond which driving ability is clearly impaired. This is due not only to the relative "crudeness" of the instrument, but also to the fact that multiple skills are required in driving. Mildly demented individuals may do well under nondemanding conditions along familiar roads, but may not adapt well to new or emergency situations. Despite these limitations, the MMSE at least provides a rough estimate of cognitive ability. Of course, if cognitive ability is found to be impaired, potentially treatable contributors (delirium, B_{12} deficiency, alcohol, thyroid abnormalities) should be evaluated and corrected if possible.

Among the changes in motor ability that occur with aging are decreasing muscle strength and joint and tendon flexibility, while muscle endurance is essentially unchanged.[26]

A directed physical examination to evaluate motor ability should include assessment of strength (manual muscle testing), range of motion, sitting balance, and proprioception. If deficits are found, efforts should be made to correct the underlying cause if possible and rehabilitation should be encouraged.

Reaction time is a performance measure that integrates elements of visual, cognitive, and motor ability. Reaction time (or perception-response time) may be defined as the time from presentation of a stimulus until the completion of a motor response to that stimulus. Despite the attractiveness of a composite measure, there are several drawbacks to using reaction time as an estimation of driving ability. First, reaction time is difficult to measure accu-

UNIVERSITY OF MARYLAND BALTIMORE

rately in an outpatient setting without specialized equipment. Second, numerous factors influence performance other than age itself, including attention (the ability to filter out unnecessary information), preparation time (the time interval between presentation of stimuli), and practice (the more familiar a task, the better the performance).[26] And finally, the relationship between reaction time performance and driving ability is not clear. Olson and Sivak compared younger and older drivers in a real world setting and found relatively little difference in reaction time.[27]

The discussion thus far has centered on patient deficits that may affect driving ability and driving safety. Clearly other factors enter into the equation as well. The Haddon injury matrix is a useful way of thinking about the different components that affect safety.[26,28-33] This approach compares the individual, vehicular, and environmental factors that are involved at different times in a crash (pre, intra-, and post-crash phases).

The vehicle plays an important role in safety. Proper maintenance is essential to ensure optimal performance. For older individuals especially, the ergonomics of the interior should facilitate operation. It should be easy to get in and out of the car and to find a comfortable seating position that ensures good outward visibility. Mirrors should be accessible and easily adjustable. Controls should be easy to reach and operate. Figures on the instrument panel should be legible and easily visible. Certain technological advances may help avoid crashes, such as antilock brakes and traction control. In the event that a crash occurs, the structural integrity of a vehicle is paramount. Safety features such as crumple zones, side impact protection, and seat back integrity help to minimize the risk of injury. Passenger restraints are perhaps the single most important element of the safety equation, since they keep the individual in the car and minimize contact with internal structures (steering wheel, dashboard, windshield). All occupants should wear seat belts at all times. Air bags may offer additional protection.

The final element of the safety equation is the driving environment. Roads should be engineered for suboptimal conditions and driver performance. Fixed roadside obstacles (trees, concrete embankments) should be avoided and break-away utility poles utilized. Speed limits (maximum and minimum) should be enforced. Low-glare lighting should be employed in roadway lights and car headlights. High-traction surfaces should be used when roads are repaved. Visible lane markings aid orientation, especially at night and when raining or snowing. Legible signs and advance warnings of intersections allow the older driver time to prepare. These factors can be phased in as new roads are built or old roads redone.

This discussion has focussed on those elements, vehicular, environmental, and individual, that may impair safety of the older driver. Driving an element of daily function; along with other means of transportation, driving is integral to the mobility of older individuals. Driving plays a special role in the lives of older men and women since they grew up with the automobile and, to many, driving is part of freedom and independence. For the physician to raise this topic and suggest the possibility of limiting or stopping driving may threaten the doctor-patient relationship. Yet there may be a conflict between safety, well-being, and lifestyle. Balancing individual and public safety against autonomy and independence is a difficult task, not made easier by the lack of reliable criteria for evaluating the effect of diseases or conditions on those functions related to driving. Further complicating the issue are the uncertain roles of the physician and the government in this process.

At present, there are no hard and fast rules about which individual factors are most closely related to driving safety, and further research needs to be done to simplify the evaluation process. Physicians should include driving issues as part of their functional assessment of older patients. Have there been problems with driving, such as getting lost or having moving violations or crashes?They should assess the elements of visual, cognitive, and motor ability discussed above. If impairments are detected that may affect driving ability and if these cannot be corrected, driving cessation or limitations may be indicated. There are a number of ways physicians can address this difficult decision. The physician can discuss the findings and implications with the patient and family; if this is not sufficient to convince a patient or family to limit or stop driving, patients may be referred to the Department of Motor Vehicles for retesting or to a rehabilitation facility for evaluation by a therapist trained to detect deficits in driving ability. The physician may detect conditions or impairments that may affect driving ability, but it is the role of a trained therapist or a government testing official to determine the actual implications of these conditions and impairments on driving ability. Both the detection of the impairment and the assessment of the effect on driving ability are essential components of the process. Some physicians may feel uncomfortable referring patients to a state agency, in which case it may be preferable to refer the patient to a trained therapist or to a geriatric assessment center where someone else can make the actual recommendation regarding driving. Even this may not be the end of the story since some men and women may continue to drive after their license has been revoked.[34] If patients do curtail or stop driving, plans must be made for alternative sources of transportation, which may be difficult if no friends or relatives are available to provide the service. Local transportation resources may be found by contacting the town government or Infoline. The entire process is awkward and cumbersome, but it represents a reasonable compromise between safety and autonomy until further research is done that allows a more rapid and accurate determination of an individual's risk for unsafe driving.

REFERENCES

1. Waller PF: The older driver. *Human Factors* 1991; 33:499-505.
2. Meier B: Safety and the older driver: When difficult issues collide. *The New York Times*, 4 May 1992.
3. Marottoli RA, Ostfeld AM, Merrill SS, et al: Driving cessation and changes in mileage among older individuals (in press).
4. Evans L: Older driver involvement in fatal and severe traffic crashes. *J Gerontol* 1988; 43:S186-93.
5. Williams AF, Carsten O: Driver age and crash involvement. *Am J Public Health* 1989; 79:326-7.
6. Evans L: Risk of fatality from physical trauma versus sex and age. *J Trauma* 1988; 28:368-78.
7. Brody JA, Brock DB, Williams TF: Trends in the health of the elderly population. *Ann Rev Public Health* 1987; 8:211-34.
8. Barancik JI, Chatterjee BF, Greene-Cradden YC, et al: Motor vehicle trauma in Northeastern Ohio. I: Incidence and outcome by age, sex, and road-use category. *Am J Epidemiol* 1986; 123:846-61.
9. Fife DF, Barancik JI, Chatterjee BF: Northeastern Ohio Trauma Study: II. Injury rates by age, sex, and cause. *Am J Public Health* 1984; 74:473-8.
10. Doege TC, Engelberg AL, eds: *Medical Conditions Affecting Drivers*. Chicago, American Medical Association; 1986.
11. *Physicians Guide to Driver Examination*. 5th ed. Ottawa: Canadian Medical Association, 1991.
12. Raffle AB, ed: *Medical Aspects of Fitness to Drive*. 3rd ed. London: Medical Commission on Accident Prevention; 1976.
13. The National Committee for Injury Prevention and Control. *Injury Prevention: Meeting the Challenge*. New York; Oxford University Press; 1989, p. 119.
14. Waller JA: Research and other issues concerning effects of medical conditions on elderly drivers. *Human Factors* 1992; 34:3-15.
15. Ray WA, Gurwitz J, Decker MD, et al: Medications and the safety of the older driver: Is there a basis for concern? *Human Factors* 1992; 34:33-47.
16. Klein R: Age-related eye disease, visual impairment, and driving in the elderly. *Human Factors* 1991; 33:521-5.
17. Shinar D, Schieber F: Visual requirements for safety and mobility of older drivers. *Human Factors* 1991; 33:507-19.
18. Johnson CA, Keltner JL: Incidence of visual field loss in 20,000 eyes and its relationship to driving performance. *Arch Ophthalmol* 1983; 101:371-5.
19. Retchin SM, Cox J, Fox M, et al: Performance-based measurements among elderly drivers and nondrivers. *J Am Geriatr Soc* 1988; 36:813-9.
20. Sivak M, Olson P, Pastalan L: Effects of driver's age on nighttime legibility of highway signs. *Human Factors* 1981; 23:59-64.
21. Keltner JL Johnson CA: Visual function, driving safety, and the elderly. *Ophthalmology* 1987; 94:1180-88.
22. Lucas-Blaustein MJ, Fillip L, Dungan C, et al: Driving in patients with dementia. *J Am Geriatr Soc* 1988; 36:1087-91.
23. Gilley DW, Wilson RS, Bennett DA, et al: Cessation of driving and unsafe motor vehicle operation by dementia patients. *Arch Intern Med* 1991;151:941-6.
24. Friedland RP, Koss E, Kumar A, et al: Motor vehicle crashes in dementia of the Alzheimer's type. *Ann Neurol* 1988; 24:782-6.
25. Folstein MF, Folstein SE, McHugh PR: Mini mental state—A practical method for grading the cognitive state of patients for the clinician. *J Psych Res* 1975; 12:189-98.
26. Marottoli RA, Drickamer MA: Psychomotor mobility and the elderly driver. *Clin Geriatr Med* (in press).
27. Olson PL, Sivak M: Perception—response time to unexpected roadway hazards. *Human Factors* 1986; 28:91-6.
28. Haddon W: A logical framework for categorizing highway safety phenomena and activity. *J Trauma* 1972;12:197-209.
29. Haddon W: The changing approach to the epidemiology, prevention, and amelioration of trauma: The transition to approaches etiologically rather than descriptively based. *Am J Public Health* 1968; 58:1431-8.
30. Haddon W: Reducing the damage of motor vehicle use. *Tech Rev* 1975; 77:1-9.
31. Haddon W: Advances in the epidemiology of injuries as a basis for public policy. *Pub Health Rep* 1980; 95:411-21.
32. Haddon W, Baker SP: Injury control. In D. Clark and B. MacMahon, eds: *Preventive and Community Medicine*, Boston: Little Brown; 1981: 109-40.
33. Robertson LS: *Injury Epidemiology*. New York: Oxlord University Press; 1992:3-22, 147-70.
34. Graca JL: Driving and aging. *Clin Geriatr Med* 1986; 2:577-89.

Assessment of Immobility in the Elderly

LEO M. COONEY, JR., M.D.

Introduction

THE quality and dignity of an older person's life often depends on the ability to maintain independence. One's personal and social independence requires the ability consistently and safety to navigate in one's own environment, as well as in the social environment in which one lives. Personal independence requires the ability to go from bed to chair, chair to toilet, and back again in a safe manner without assistance. Social independence requires such abilities as climbing stairs, transferring in and out of an automobile without help, or living in an environment which allows appropriate handicapped access. While altered mental status and loss of bladder and bowel continence are frequent causes of dependence and subsequent nursing home admissions, loss of mobility is often the most remediable cause of loss of independence. Aggressive evaluation and treatment of immobility can often prevent nursing home admissions and promote a dignified, independent, life-style.

There are many factors which affect an individual's ability to be safely mobile in his or her own environment. Alterations in the musculoskeletal, neurologic, cardiovascular, and sensory systems can impair mobility. Mobility can be lost because of severe systemic illness, forced immobility, medications, depression, loss of motivation, and fear of falls and injury.[1] An assessment of mobility requires that the physician has determined whether the patient's problem is due to systemic disease or weakness, or due to a local musculoskeletal or neurologic problem. The first step in this determination is a careful observation of the patient's attempt at transfers and ambulation, followed by a focused physical examination.

Personal Mobility

Personal mobility includes the ability to turn in bed independently, to go from supine to sitting, sitting to standing, bed to chair, or chair to toilet by oneself, to walk or propel oneself in a wheel chair without assistance, to climb stairs independently, and to navigate one's environment safely.

A combined assessment by a physical or occupational therapist and a physician can be an effective method of identifying and treating the causes of immobility. The therapist, after assessing all components of mobility, will identify that maneuver which most limits independence. The physician and therapist together can then determine what underlying process or processes are the cause of the difficulty with this maneuver. Once the "rate-limiting" factor to full mobility is determined, an approach to therapy can be developed with such techniques as medications, local steroid injections, bracing, surgery, or physical rehabilitation.

Mobility begins with the capacity to turn in bed independently. It is difficult to care for individuals at home who cannot turn by themselves. Bed immobility puts patients at risk for pressure sores, flexion contractures of joints, and aspiration pneumonia. Patients with profound dementia often do not turn themselves independently; the physical problems most frequently associated with bed immobility are dense hemiparesis, profound muscle weakness from such conditions as polymyositis, and morbid obesity.

Transfers

The maneuver of moving into and out of a chair or bed is termed a transfer. Difficulty with transfers is a common problem of older individuals, affecting 6% to 8% of those over 65.[3] Inability to transfer is a frequent cause of nursing home admissions, and is the single most impor-

---

LEO M. COONEY, JR., M.D., Director, Program in Geriatrics, Humana Foundation, Professor of Geriatric Medicine, Professor of Medicine, Yale University School of Medicine, New Haven.

tant maneuver in the maintenance of physical independence.[4]

The first step in any transfer from bed to chair is the move from supine to sitting. This can be a difficult task for patients with a dense upper extremity hemiparesis, severe arthritis affecting the wrists, elbows, or shoulders, and other causes of upper extremity weakness or pain. The patient must be able to roll in bed independently to do a supine to sit transfer, and usually needs at least one-sided good upper extremity function, as an older individual must often push or pull into a sitting position. Hospitalized patients often use such special apparatus as a trapeze hung over the bed, but these assistive devices are less often available at home. It is difficult for patients with casts on their lower extremities to do a supine to sit transfer independently. Upper extremity joint pain can greatly limit this maneuver. Judicious management of upper extremity weakness or pain can significantly improve supine to sit transfers.

Alexander et al demonstrated that older patients had more difficulty going from supine to sit than younger controls, and usually had to use their upper extremities or legs to assist with a supine to sit transfer.[5] Younger patients could often do this transfer with no use of their arms. This reliance on upper extremity assistance makes it difficult for older patients to go from supine to sitting when they have upper extremity weakness or pain.

The sit-to-stand or sit-to-sit transfer is the most important maneuver for physical independence. One cannot be left without human assistance for a extended periods of time if one cannot get from bed to toilet and toilet to chair or bed independently. Strength, balance, and good safety awareness are essential in order to perform these transfers safety and effectively.

Observation of Transfer

Direct observation of a patient attempting a transfer is the best way to identify those conditions that impede this maneuver. Does the patient swing her upper body back and forth building up to the transfer? Patients with immobile hips and knees, weak knee extensors and hip extensors, and difficulty with fully extending their trunk often initiate transfers with this maneuver. Does the patient push or pull himself up, bearing a great deal of weight on the upper extremities? This maneuver indicates either weakness, immobility, or pain in the lower extremities. Does the patient bear full weight on her legs when transferring? The inability to bear full weight usually indicates a painful condition affecting foot, ankle, knee, hip, or leg bones. Does the patient fully flex and extend his knees when transferring? The inability to flex the knee to a 90° angle impedes transfers. The patient must thrust his weight further forward if he cannot fully flex the knees. Patients with a fused knee require excellent flexibility and strength of the contralateral knee to do a successful sit to stand transfer. Can the patient fully extend his knee? Knee flexion contractures prevent an individual from standing straight, and thus impede balance, transfer of weight, and patient flexibility with transfers. Can the patient fully flex and extend his or her hips? Lack of full hip flexion prevents the patient from sitting at a 90° angle. It is more difficult to go from sitting to standing if one cannot bring the trunk forward to 90°. Lack of full hip extension also prevents a patient from standing straight, thus impeding appropriate weight shifting, and balance.

Types of Transfers

There are four common methods of getting from a bed to a chair or chair to toilet. One can do a stand-pivot transfer, an anterior-posterior transfer, a side-to-side transfer, or can transfer with the assistance of a sliding board.

Good “static sitting balance” is an essential prerequisite to a successful transfer. The patient must be able to sit unsupported before starting either a sit-to-stand or a sit-to-sit transfer. Truncal stability may be affected by such neuromuscular diseases as strokes, multiple sclerosis, Parkinson’s disease, or midline cerebellar degeneration.

Once the patient has achieved good static sitting balance in rehabilitation from such conditions as strokes, he must be able to maintain truncal stability when shifting weight to perform a transfer. This ability is termed “dynamic sitting balance.”

A stand-pivot transfer requires going from a sitting to a standing position, often with the assistance of a walker, and then turning or pivoting to a chair or toilet. In order to do a successful stand-pivot transfer, the patient needs stable, mobile lower extremities with little or no pain on weight bearing, and needs to be able to bear full weight on at least one upper extremity. The patient must be able to push off the bed or chair with upper extremities if necessary.

Patients who lack part of one or both lower extremities due to amputations, who are unable to bear full weight on lower extremities due to weakness, pain, or a recent fracture, or who lack enough strength or mobility of their lower extremities to do a stand-pivot transfer, can often be taught to transfer safely using a sliding board. These transfers require a level surface between bed and chair, or chair and toilet. The patient must have excellent static and dynamic sitting balance, and must be able to lift his body weight with the upper extremities. This maneuver requires careful attention to detail and excellent safety awareness.

Patients with excellent balance and upper extremity strength, but weak or absent lower extremities, can sometimes swing from bed to chair or chair to chair in an anterior/posterior transfer or side-to-side transfer without a sliding board. This maneuver is frequently used by younger individuals with traumatic amputations or paraplegia. It is rare that an older patient will have enough upper extremity strength and balance to lift all of his weight on the upper extremities and swing that weight the distance from a bed to chair without a sliding board.

Safety Awareness

Therapists often use the term "safety awareness" in describingpatients' ability to know their limits with mobility. A patient with good safety awareness will know when and when not to use an assistive device, and when to wait for help with a particular maneuver; he will consistently lock his wheel chair, ensure that devices are properly positioned before starting a maneuver, and recognize his limitations when weak or ill. Safety awareness can be a major problem with older patients with dementia, delirium, or other alterations in mental status. In addition, patients with such central nervous system problems as strokes and tumors often have perceptual and cognitive deficits, impulsivity, and one sided neglect. Patients who do not know their limitations cannot be safely left alone.

Ambulation

While an individual can be totally independent with a wheel chair, use of this device can be a problem in homes or apartments with stairs, narrow doorways, small bathrooms, and in kitchens with high shelves, sinks, stoves, and other appliances. Full independence with a wheel chair usually requires a specially adapted environment. In addition, reliance on a wheel chair for mobility can result in lower extremity muscle weakness and decreased mobility of the hips and knees. Independence in walking, therefore, can greatly improve the functional abilities of most older individuals.

The ability to walk in an independent and functional manner requires adequate strength, balance, and the ability to bear weight in a sequential fashion. One must have enough strength to stabilize the hip, have antigravity hip flexion and abduction, and stability of knees and ankles in order to walk independently. Balance is essential to safe and effective ambulation; the joints of the lower extremities must allow the individual to bear weight equally and sequentially.

Gait Patterns

Patients with difficulty in walking should be closely observed as they walk: a normal gait requires that an equal amount of time be spent on each lower extremity. As one walks, the heel should touch the ground first, followed by the mid-stance phase in which the weight is distributed from heel towards the front of the foot, followed by the "push off" phase in which one pushes weight off the "balls" or metatarsal-phalangeal joints of the foot. Pain in any part of the lower extremity, either in the bones of the lower extremity or in the joints of the foot, ankle, knee, or hip, will result in less time weight bearing on the affected side, producing an antalgic gait or limp. If the cause for pain is at the ankle or above, the individual spends less time in the "mid-stance" phase of walking, when the weight is shifted forward from the heel to the front of the foot. If the cause for pain involves the foot itself, the patient will bear weight in an abnormal fashion to avoid putting pressure on the part of the foot that is painful.

In walking, the stride should be equal in each leg and two to three feet in length. Each leg should lead forward in a sequential fashion; individuals who consistently lead with one leg may have weakness on one side or significant hip disease on that side. Patients with normal neuromuscular function usually swing their arms in a symmetric manner as they walk. Close observation of upper extremity movement may indicate hemiparesis or other neurologic problems.

If the stride is noticeably shorter than normal, one should assess the individual for hip disease, as full flexion and extension of the hip is necessary for a full stride. Other conditions that produce short strides are Parkinson's disease, flexion deformities of the spine, and balance problems.

Patients with localized muscle weakness have characteristic gait patterns. Patients with ankle dorsiflexion weakness due to a peroneal palsy, L4-L5 disease, or brain lesions, often walk with a "hitched" gait, in which they hyperflex the hip in order to clear the ground with the toe of the foot; these patients may also drag the affected foot. Patients with hip abduction weakness often have a "lurching" gait, in which they side flex their trunk to the side opposite to the weakness. Patients with hip extensor weakness often hyperextend the trunk as they walk.

Patients with decreased proprioception, due to such conditions as a peripheral neuropathy or posterior column disease, have a characteristically wide-based "slapping" gait in which the foot appears to be searching for the ground. Patients with normal pressure hydrocephalus have a gait pattern in which the foot does not move far from the ground, and are often described as having "feet glued to the ground."

Balance is extraordinarily important to safe ambulation. Static truncal instability is seen in patients with cerebellar disease, advanced Parkinson's disease, and demyelinating diseases such as multiple sclerosis. Truncal instability with movement is often seen with patients with hemiparesis, movement disorders such as chorea, and vestibular disease.

Performance Tests of Ambulation and Falls

Tinetti has proposed an evaluation of a patient's ability to ambulate safely.[8] The patient rises from a firm armless chair and stands, feet close together, for 30 seconds. If the patient becomes light headed, pulse and blood pressure are obtained. A firm tug on the patient's waistband from behind determines if the patient can maintain steadiness when upright. The examiner then watches the patient walk across a room and observes for characteristic gait abnormalities. The patient then turns 180° and sits down in a chair. Individuals who can consistently perform these maneuvers have little risk of falling.

Two other functional tests are helpful in determining mobility performance. These are the stair-climbing test, in which the patient climbs five stairs and the examiner makes note of fatigue, instability, and the patient's technique, and the walking endurance test, in which the total distance covered in five minutes is measured.

Watching a patient turn can identify a number of problems, and help determine which patients are at high risk for falling. Vestibular disease can make turning difficult, and falls occur frequently when patients look up and turn at the same time. Patients who have pain on weight-bearing on one leg may lose their balance and fall when they shift weight to the painful leg during a turn. The synchronous movement of a turn is often the first physical maneuver lost with early Parkinson's disease. Close observation of the turn of a patient with early Parkinson's disease will demonstrate that the patient takes several steps, not one smooth step, during this maneuver.

Determining Causes of Immobility

The overall assessment of mobility problems requires a dynamic assessment of a patient's ability to transfer and walk in a real environment. This assessment must begin with close observation of the patient going through all phases of mobility. Once this observation has determined the rate limiting factors to independent mobility, a thorough physical examination focused on that phase of mobility must be done. This should include a full examination of the joints and a manual muscle examination to determine the extent and pattern of any muscle weakness. The use of diagnostic imaging and laboratory techniques should be used only to complement these observations. These tests should not be ordered until the observation and physical examination has identified the most likely causes of limitations to mobility.

A patient may have difficulty going from the sitting to standing position because of decreased extension of the hip. Osteoarthritis of the hip joint is the most common cause for loss of this movement. Patients with myopathies, such as polymyositis, steroid myopathy, or rhabdomyolysis, have symmetric proximal muscle weakness, most pronounced in the lower extremity; the hip girdle muscles are thus the most affected. Neurologic diseases cause more distal than proximal weakness, but may contribute to hip problems. The gluteus maximus muscle, responsible for hip extension, is innervated by L5-S1 and S2, and can be affected in lumbar disc disease or lumbar spinal stenosis. The inferior gluteal nerve, which innervates the gluteus maximus muscle, can be impaired in diabetic neuropathy, producing the so-called "diabetic amyotrophy." In addition to the gluteus maximus muscle, the "hamstring" muscles behind the knee, including the semitendinosus, semimembranosus, and the long head of the biceps femoris, are involved in hip extension. All three of these muscles receive their innervation from the sciatic nerve and nerve root innervation from primarily L5-Sl and S2. Impairment of the sciatic nerve can produce weakness in these muscles. Hip function can also be limited because of inflammatory arthritis, hip flexion contractures due to prolonged immobility such as confinement to a wheelchair, pain around the hip due to trochanteric bursitis, and the pain of severe sciatica.

Knee extension is a key component of a sit-to-stand transfer. Arthritis of the knee, especially the patellofemoral component, is a frequent cause of difficulty with this maneuver. Patients with severe and long standing arthritis of the knees often develop knee flexion contractures, and lose the ability to fully extend their knees. The quadriceps muscle extends the knee; this muscle gets its nerve root innervation from L2, L3, L4, and its peripheral innervation from the femoral nerve. Femoral neuropathies, often seen in diabetes, will produce weakness of both hip flexion and knee extension. Quadriceps atrophy is often produced by knee disease. Knee effusions impair an individual's ability to maintain and improve quadriceps strength. Prolonged bed rest, confinement to a wheel chair, and lower extremity injuries also cause quadriceps atrophy and weakness.

Ascending and descending stairs requires a combination of good balance, good strength, and good function of both the hips and knees. Ascending stairs requires good hip flexion and knee extension. Patients with significant hip disease have most difficulty ascending stairs, as this maneuver requires the most movement and flexion of the hip joint. The muscles responsible for hip flexion are the psoas major and the iliacus. These muscles both obtain their nerve root innervation from the second and third lumbar nerve, and their peripheral innervation from the femoral nerve. Hip flexion is limited with both hip disease and significant muscle disease. Knee extension is required to push off one step and pull the contralateral leg up to the next step. The causes of knee extension weakness outlined above can make this maneuver difficult.

Descending stairs puts less pressure on the hip joints and muscles, but a great deal of pressure on the knee. Descending stairs requires full weight on one knee in the flexed position. This maneuver puts a great deal of stress on the patellofemoral component of the knee joint. Patients with osteoarthritis of the knee or "chondromalacia patellae" can have great difficulty with this maneuver. Patients with osteoarthritis of the patellofemoral component of the knee often descend steps one at a time, or backward, to avoid putting weight on the flexed knee.

Bracing

The stability of joints and lower extremities can be improved with bracing. It is difficult, however, to stabilize knee joints fully if significant arthritis is present. Orthopedic knee braces may be helpful for patients with structural damage to a meniscus or collateral ligament, but rarely improve function in patients with inflammatory arthritis or osteoarthritis. Long leg braces, which allow patients with antigravity hip flexion strength but lower extremity weakness to stand erect and walk, are often heavy and cumbersome. They have a major role in younger patients with spinal cord injuries or other causes of paraplegia or hemiplegia, but are less frequently helpful for older patients.

Short leg braces can be very helpful in the rehabilitation of patients with neuromuscular conditions. These braces include a plastic ankle foot orthosis (AFO) which can be inserted into the shoe, or external metal braces attached to the heel and sole of the shoe. These braces keep the foot in a dorsiflexed position and prevent foot dragging or hyperflexion of the hip in patients with a foot drop. In addition, these braces can stabilize the hind foot or subtalar joint, and can also prevent hyperextension of the knee, or genu recurvatum, by preventing the angle between the lower leg and foot from exceeding 90°. This hyperextension can damage the knee joint and occurs when patients with profound quadriceps weakness attempt to walk in an unsupported manner.

Assistive Devices

An assistive device can make walking easier and safer by decreasing the weight borne on a painful extremity and by stabilizing a patient's gait pattern. The selection of an appropriate device must be done individually after a complete evaluation by a physical therapist. The factors that affect this selection are beyond the scope of this review, but there are several concepts which might be helpful.

A standard walker gives the most stability for walking, but requires bilateral upper extremity function and the ability to pick the walker up and lift it forward. A standard walker may not fit in a small toilet, and cannot be used to climb stairs.

A hemiwalker is a wide-based, stand-alone device that can be helpful in distributing and balancing the weight of a patient with a dense hemiparesis, and who cannot use a standard walker.

Crutches are helpful for patients with orthopedic problems. They allow ambulation with little or no weight-bearing on one leg. They do put a great deal of pressure on the wrists and shoulders, and require excellent balance. They allow stair climbing and maneuvering in close quarters, but must be used cautiously by older patients with upper extremity difficulty and balance problems.

A "quad cane" is a cane with a four-pronged base which stands alone and gives more stability than a standard cane. Patients need to be instructed carefully and monitored closely in the use of canes. A cane can redistribute up to 40% of the lower extremity body weight off a painful hip or knee, but must be used correctly. The cane should be carried on the side opposite the painful lower extremity; the cane must be placed on the ground simultaneously with the painful leg, to distribute the weight properly away from this extremity.

Conclusion

The cause of a patient's difficulty moving in his environment can be determined by a careful analysis of a patient's mobility. Observation is the key to this analysis. Watching the patient carefully as he rolls over in bed, goes from a supine to sitting position, transfers from sitting to standing, walks, turns, and ascends and descends stairs is the essential first step in the analysis of mobility problems. A focused joint, muscle, and neurologic examination, directed to that region which appears to be limiting mobility, will then lead to the appropriate diagnostic imaging and laboratory studies. This stepwise approach can lead to the most appropriate medical, surgical, and rehabilitative therapy, and ensures that such therapy will have a major affect on a patient's ability to function more independently.

REFERENCES

1. Ettinger W: Immobility in the elderly. In: Kelley,W, ed. *Textbook of Internal Medicine*. Philadelphia: JP Lippincott; 1992: 2,400.
2. Leon J, Lair T: Functional status of the noninstitutionalized elderly: Estimates of ADL and IADL difficulties (DHHS

Publication No. (PHS) 90-3462). National Medical Expenditure Survey Research Findings 4, Agency for Health Care Policy and Research. Rockville, Md: Public Health Service.

3. Dawson D, Hendershot G, Fulton J: National Center for Health Statistics. Aging in the eighties. Functional limitations of individuals age 65 and over. Advance Data from Vital and Health Statistics. No. 133. (DHHS Pub. No. 87-1250). Public Health Service, Hyattsville, Md, 10 June 1987.
4. Branch LG, Meyers AR: Assessing physical function in the elderly. *Clin Geriatr Med* 1987; 3:29-51.
5. Alexander NB, Fry-Welch DK, Ward ME, et al: Quantitative assessment of bed rise difficulty in young and elderly women. *J Am Geriatr Soc* 1992; 40:685-91.
6. Alexander NB, Schultz AB, Warwick DN: Rising from a chair: Effects of age and functional ability on performance biomechanics. *J Gerontol* 1991; 46:M91-8.
7. Barbenel JC, Ferguson-Pell MW, Kennedy R: Mobility of elderly patients in bed: Measurement and association with patient condition. *J Am Geriatr Soc* 1986; 34:6336.
8. Tinetti ME. Performance-oriented assessment of mobility problems in elderly patients. *J Am Geriatr Soc* 1986: 34:119.
9. Wolfson LI, Whipple R, Amerman P, et al: Stressing the postural response: A quantitative method for testing balance. *J Am Geriatr Soc* 1986; 34:845.

Opportunity Hotline

Taking the Search out of Physician Search

Direct, confidential referral to
Family Practice and OB/GYN
practice opportunities NATIONWIDE.

Call us toll free and within a
few weeks you will be contacted
directly by hospitals, clinics,
or doctors groups who know your
practice and geographic preferences
and are interested in YOU.

It's that simple and no recruiters
are involved with this service.

800-264-4456

A Right to Health Care Providers Background on Whether Americans are Entitled to Receive Health Care Services

Although not legally established, the idea that Americans have a right to some level of health care has been widely discussed for many years in literature about fair access to health benefits.

The National Reference Center for Bioethics Literature has issued a new paper, *A Right to Health Care*, Number 20 in its Scope Note Series, which provides a brief essay discussing the background of the debate and 15 pages of annotated bibliographic citations to literature concerned with whether or not health care should and can be provided to all. Certain groups in the United States are already the recipients of government funded health services, and much has been written about extending these services to all citizens.

The Scope Note Series offers an overview to topics in biomedical ethics and provides immediate reference to facts, opinions, or legal precedents (if applicable). Useful to medical and legal practitioners, scholars, journalists, students, and interested laypersons, Scope Notes are available from the National Reference Center for Bioethics Literature, Kennedy Institute of Ethics, Georgetown University, Washington, DC 20057-1095, for $5.00 each, prepaid ($8.00 for overseas airmail).

A Right to Health Care was published in the Kennedy Institute of Ethics Journal 2(4): 389-405, December 1992.

UNIVERSITY OF MARYLAND AT BALTIMORE

Stroke Prevention in the Elderly

RICHARD W. BESDINE, M.D.

ABSTRACT—Stroke is a common, devastating and frightening event among older persons, often resulting in death or major loss of independence. Professional and lay attitudes of the past have assumed an inevitable and immutable course of stroke risk progression with age. Although stroke is strongly age-associated, recent data have accumulated demonstrating reduction in stroke rate by preventive interventions in older patients with risk factors. Statistically significant stroke reduction has been achieved by 1) antihypertensive treatment of patients with elevated systolic blood pressure, even beyond age 80; 2) anticoagulation with warfarin for patients with chronic or intermittent non-valvular atrial fibrillation; 3) use of low-dose aspirin in patients with TIA or nondisabling stroke; and 4) carotid endarterectomy for patients with symptions and ipsilateral serve stenosis. Clinical strategies to prevent stroke in older adults are now tested and proven useful; they should be used as indicated in older persons at risk.

Introduction

STROKE has been defined as the sudden or rapid onset of focal neurologic deficit(s) lasting longer than 24 hours; transient ischemic attack (TIA) lasts less than 24 hours. Many stroke experts have more recently, for definitional purposes, limited the duration of deficit in TIA to six hours or less. The reversible ischemic neurologic deficit (RIND) written about in the 1970s and 1980s is no longer considered a separate entity; based upon more sophisticated and detailed imaging studies, it is, believed to be a small stroke with rapid recovery of function.

In 1992 there were one-half million strokes in the United States; 20%, or 100,000 persons, died within one month of the event, and another 10% died or will die within one year. Of the survivors, 35,000 are severely disabled (requiring institutional care), and another 100,000 will be able to remain home with less severe residual deficits. Older persons bear the brunt of stroke mortality and morbidity; 75% of strokes and nearly 90% of accompanying deaths occur in older individuals. These data[1] underscore the importance of stroke to Americans in spite of a gratifying downward trend of stroke and stroke mortality over the past 40 years.[2,3,4] Recent reports of a leveling off or even an increase of stroke incidence have been verified, but can be explained by our increased capacity to detect small strokes by using contrast-enhanced computerized tomography of the head and magnetic resonance imaging during the decade of the 1980s.

In spite of these reassuring observations, stroke is nonetheless of great concern; it remains the third leading cause of death over age 65 and is number two, behind heart disease, over age 85.[5] Stroke is the fourth most common cause of disability among older persons, and the disabilities it produces tend to be severe. Most physicians and the public take stroke seriously and are most interested in opportunities for prevention rather than in strategies to treat stroke. Recent literature has been replete with reports of reduction of risk for stroke by four different health care interventions: antihypertensive therapy of systolic hypertension; aspirin; anticoagulation in persons with nonvalvular atrial fibrillation; and carotid endarterectomy in symptomatic patients with ipsilateral severe carotid narrowing.

Isolated systolic hypertension is a major risk factor for stroke, cardiac morbidity and mortality, and overall mortality; risk increases with age. Elevated systolic high blood pressure can be lowered safely. But what was not known until last year was whether lowering isolated systolic hypertension would reduce an older person's risk

---

RICHARD W. BESDINE, M.D., Professor of Medicine, Director, Travelers Center on Aging, Travelers Professor of Geriatrics and Gerontology, University of Connecticut School of Medicine, Farmington.

of stroke, heart disease, and death. Recent studies have also called into question the past practice of not giving anticoagulants to older patients with nonvalvular atrial fibrillation, have shed more light on the benefits of aspirin therapy, and have reaffirmed the value of carotid endarterectomy as a preventive measure in high-risk symptomatic patients.

Prevention of Stroke by Antihypertensive Treatment of Isolated Systolic Hypertension

Seven years ago the National Institutes of Health launched a major prospective trial designed to look specifically at the effect of isolated systolic hypertension (ISH) in older people. ISH increases risk for stroke in older adults by 50-500%, depending upon age, associated risks, and duration of follow-up.[1-6] The Systolic Hypertension in the Elderly Program (SHEP) has now issued its final report, a landmark publication in the field of stroke prevention.[7]

UNIVERSITY OF MARYLAND AT BALTIMORE

The SHEP study enrolled nearly 5,000 community-dwelling subjects over the age of 60 from 18 clinical centers, all of whom had systolic blood pressures ranging from 160 to 219 mm Hg and diastolic pressures of less than 90 mm Hg. Participants were randomly assigned to a placebo group or to treatment, which proceeded stepwise, beginning with 12.5 mg a day of chlorthalidone (CLD). The CLD dose was doubled for those who did not meet the target blood pressure goal of 140 mm Hg or a reduction of at least 20 mm Hg at follow-up visits. When CLD failed to achieve that goal, atenolol (ATL) was added to the regimen, first at 25 and then at 50 mg a day.

Diuretic therapy alone reduced blood pressure to goal in nearly half the treated patients, and more than two-thirds reached goal with combinations of CLD and ATL. Systolic blood pressure was lowered an average of 26 mm Hg and diastolic pressure an average of 9 mm Hg in the treatment group. Systolic and diastolic blood pressure lowering of 14 and 4 mm Hg, respectively, also occurred in the placebo group. Although placebo effects are to be expected, more than a third of placebo subjects were eventually treated for ethical reasons, so some of their reduction reflects both a therapeutic and placebo effect.

The events analyzed in the five-year follow-up period included nonfatal and fatal strokes, cardiovascular and coronary morbidity and mortality, all-cause mortality, and quality of life. For the first time, treatment of ISH in older adults in a rigorous randomized controlled trial was proven to reduce stroke and coronary disease, even including subjects with known cardio- and cerebrovascular disease and diabetes. During the five years, 30 strokes were prevented for every 1,000 persons treated and major cardiac events were reduced by 55 per 1,000.

Because analysis was performed by intention-to-treat, placebo subjects who were eventually treated on ethical grounds were nevertheless included in the placebo group for statistical calculation of benefit; statistical reduction of risk was still impressive. Moreover, the study demonstrated, for the first time, the benefits of treating patients over the age of 80. Side effects were minimal and statistically not significant; quality of life was not adversely affected in the treated group. Although there was no difference in all-cause mortality between the treatment and placebo groups, there was a trend toward lower mortality with treatment.

These SHEP findings lead to firm recommendations about the treatment of ISH in older people. ISH is a deadly condition that can be treated safely and effectively to reduce stroke and coronary events. Target systolic pressure should be 140 mm Hg or at least a 20 mm Hg reduction. Reductions below 140 mm Hg are likely to be accompanied by symptoms of hypotension and inadequate organ perfusion, and are dangerous.

Until data showing that newer agents can achieve at least as good risk reduction for stroke and heart disease are collected, the preferred initial treatment is a thiazide diuretic, such as CLD or an equivalent, at a starting dose of 12.5 mg a day. Alternatives to step 1 thiazide or step 2 beta-blocker therapy have to be chosen on an individual basis. Although it is well established that older patients with hypertension have decreased cardiac output and increased peripheral resistance, and that thiazides are theoretically not a good choice because they exacerbate both these physiologic deficits, we have no data supporting the efficacy of any other drugs for reducing stroke and heart disease risk in patients with ISH. Recent studies on the effects of angiotensin-converting enzyme inhibitors on reducing the risks accompanying left ventricular dysfunction may make these the agents of choice in the future for older persons with hypertension;[8] we await studies demonstrating reduction of stroke risk with ACE inhibitors in older hypertensives. Finally, after target blood pressure has been successfully maintained for six months, dosage reduction should be attempted since requirements often diminish following effective treatment.

Prevention of Stroke in Nonvalvular Atrial Fibrillation

It had been past practice to avoid anticoagulants in older adults with nonvalvular atrial fibrillation (NVAF); embolic events were thought to be rare and anticoagulants especially dangerous. Although NVAF is rare in the fourth decade of life (0.2/1,000), rates increase rapidly with age and reach 39/1,000 over age 80; rates of 2-5% are reported over age 65. The risk for ischemic stroke is increased five-fold in the presence of NVAF, and more and more strokes are associated with NVAF as age increases (36% of first strokes over age 80).[9-10] For women over age 70, NVAF is the single risk most likely to cause stroke.

Three major recent studies that have documented the efficacy of warfarin or aspirin in preventing stroke in older patients with nonvalvular atrial fibrillation were summarized by Albers.[11] The Stroke Prevention in Atrial Fibrillation (SPAF) nation-wide trial,[12] the Boston Area Anticoagulation Trial for Atrial Fibrillation (BAATAF),[13] and the Copenhagen Atrial Fibrillation, Aspirin, Anticoagulation study (AFASAK)[14] each excluded high-risk patients, including those who had suffered an embolic event or myocardial infarction within the previous six months, those with congestive heart failure, and those with cardiomyopathy. In the SPAF study, patients under the age of 60 with atrial fibrillation were randomized only to aspirin because their stroke risk appears to be so low.

Each study showed a reduction in stroke by both intention-to-treat and on-treatment analysis. The pooled data show 69% fewer strokes—a reduction from a 4.5% average annual incidence (range 3.0-7.0%) to a 1.5% annual incidence (range 0.4-2.3%). Moreover, most of the strokes in the warfarin-treated patients occurred during a period of noncompliance or inadequate anticoagulation. The on-treatment analysis yielded a risk reduction of 83% (average annual stroke incidence of 0.8%). There was no difference in stroke rates between those subjects with chronic AF compared with those who had intermittent or paroxysmal AF.

The incidence of mild, moderate, and severe stroke was reduced equally in the studies. In each of the trials, one fatal cerebrovascular hemorrhage occurred in a warfarin-treated patient. Although any such deaths are of concern, severe bleeding was not statistically significantly more common with warfarin therapy. Minor bleeding, however, was about three times more common with warfarin.

An additional point worth noting is that the prothrombin time target range for anticoagulation in these studies was as low as 1.2 times control, or 15 to 16 seconds. Most recently, it has been suggested that *any* prolongation of prothrombin time may be more than sufficient to achieve the desired reduction in stroke risk for NVAF individuals. It would appear that for the past 20 years in the United States we have been using doses of anticoagulants that are far too high.

Aspirin significantly reduced the risk of stroke only in the SPAF trial and not in the other two, perhaps because SPAF randomized young, very low-risk patients to aspirin or placebo only; warfarin was not an option. In addition, even in SPAF, aspirin did not protect subjects over age 75, nor did it prevent severe strokes. Although the arm of the trial which randomized subjects to placebo *vs* either aspirin or warfarin was interrupted in 1990 when analysis showed that both aspirin and warfarin significantly reduced ischemic and embolic strokes, an arm of the trial that randomly assigned similar patients to warfarin or aspirin is continuing in both SPAF and AFASAK; both AFASAK and BAATAF strongly suggest that warfarin is superior to aspirin in reducing stroke risk *in subjects with AF*. In persons over age 75 with NVAF, who are at highest risk for stroke, it appears that warfarin is the only effective preventive therapy.

In late 1992 a randomized, double-blind, placebo-controlled trial of warfarin in veterans with NVAF was reported; it, as with all others, was terminated early because of clear benefit in the treated group.[15] The findings, though not different from those reported one-to-two years earlier, are reassuring. The benefit was a 70% reduction in stroke risk, using low-intensity (1.2-1.5 prothrombin time ratio) anticoagulation; there was only one intracerebral hemorrhage in the warfarin group, and major bleeding was not more common. Previous stroke was a powerful risk factor, but risk was also reduced by warfarin.

The SPAF investigators cleverly looked beyond the obvious effects of aspirin or warfarin on stroke incidence for other insights that could be gleaned from that enormous data base. Accordingly, the placebo group was analyzed separately to look for additional risk factors beyond AF that could be used to identify patients at highest risk for stroke and thus those for whom the potential hazards of warfarin therapy might be justified.[16]

Within the placebo group, the investigators found that three clinically identifiable factors predicted stroke risk increase beyond that associated with NVAF alone. Hypertension, congestive heart failure within the preceding three months, or a previous thromboembolic event was each associated with an increase in stroke risk greater than 7% a year. Those variables thus define populations with stroke rates ranging from 2.5% in those with no risk factors, to 7.2% with any one risk factor, and to 17.6% with two or three risk factors. In patients without any risks, advanced age and diabetes each was found to be additional independent risk. But no strokes at all occurred in nondiabetic patients under the age of 60 who had no clinical risk factors.

In a second paper, the findings at entry on M-mode and 2-D echocardiography were analyzed to see whether they provided any additional value in risk assessment.[17] Although clinical intuition might suggest that echocardiography would not contribute much in the face of groups with such clearly defined clinical risks, left ventricular dysfunction on 2-D and left atrial size on M-mode echocardiography proved to be strong independent predictors of ischemic stroke or embolic events. Echocardiographic data changed the classification of 18% of the subjects overall, and of 38% of the subgroup with no clinical risk factors. Clinical criteria, therefore, will identify 58% and echocardiography an additional 20% of patients at higher risk who presumably would benefit most from treatment. Echocardiographic abnormalities did not increase the risk in those who already had two or more clinical risk factors; accordingly, the study findings are likely to be most useful in those with no clinical risk factors.

Current findings clearly support treating patients with NVAF with aspirin at the very least. Although definitive recommendations cannot be made until the trials comparing aspirin and warfarin are completed, it seems very likely that older patients with NVAF and any additional risk factors, clinical or echocardiographic, would benefit most in stroke reduction from treatment with warfarin, as long as there are no major contraindications. The indications for warfarin become increasingly urgent as the risk of stroke increases. Aspirin might be sufficient for some patients with atrial fibrillation only, but keep in mind that diabetes and age over 75 or 80 each increases the risk of stroke in patients with no other risks. In addition, heart failure, hypertension, and echocardiographic abnormalities all become more common with advancing age, and all increase both the risk of stroke and the risk reduction with warfarin. Certainly it makes sense to identify clinical factors known to increase risk for stroke in those with NVAF: when none is present, echocardiography is indicated to identify any additional risk factors.

UNIVERSITY OF MARYLAND AT BALTIMORE

Prevention of Stroke by Aspirin in Previous Minor Stroke or TIA

In 1987 it was definitively demonstrated that aspirin reduced the risk of death from vascular causes and of nonfatal stroke and myocardial infarction in patients with TIA or nondisabling stroke.[18] The compiled results of many trials show a risk reduction of about 25% with high-dose aspirin therapy—one-to-four 325 mg tablets a day. Because more recent studies have shown that doses as low as 30 mg a day are as effective as high doses in stopping platelet production of thromboxane A2, which is the key to reducing platelet adhesiveness, investigators reasoned that lower doses might be as effective in reducing the incidence of vascular disease and less likely to cause adverse gastrointestinal effects.

Accordingly, the Dutch TIA Trial Study Group randomized 3,100 patients with TIA or minor stroke within the preceding three months to treatment with either 30 or 283 mg of aspirin a day.[19] After 30 months of follow-up, low-dose aspirin was found to be at least as good as high dose in preventing strokes—perhaps even better, although the 10% difference was not statistically significant. A theoretical explanation for the greater effectiveness of a lower dose does exist. Although a 30-mg dose will reduce platelet adhesiveness, it has little effect on cyclooxygenase, and thus on prostacycline production, in other cells. Consequently, the effect of prostacycline on maintaining a smooth vascular endothelium would not be diminished, and platelet adherence would not be encouraged.

Another interesting report last year came from the Swedish Aspirin Low-dose Trial (SALT).[20] That study, begun in 1987 before the benefits of usual-dose aspirin were established, compared low-dose aspirin and placebo, a study design that today would be considered ethically unacceptable. The subjects were 1,360 patients with previous TIA or minor stroke; they were randomized to 75 mg a day of aspirin or placebo and followed for 32 months. Both men and women in the treatment group had a reduction in stroke or mortality of 18%. This study is and likely will remain unique in showing the benefit of low-dose aspirin compared with placebo.

Prevention of Stroke by Carotid Endarterectomy

Enthusiasm for carotid endarterectomy to prevent stroke in patients with prior stroke or TIA, and to prevent first strokes in those with carotid stenosis, increased dramatically during the 1970s and peaked in 1985, when more than 100,000 of these procedures were done in spite of negative reports from randomized clinical trials. These negative data, added to 1) declining stroke rates over 20 years, 2) the demonstrated benefit of aspirin and other antiplatelet drugs, 3) a new emphasis in the medical community on risk reduction for stroke, and 4) a negative trial of extracranial-intracranial bypass in 1985, resulted in sharp declines in carotid endarterectomies and a growing belief that such surgery actually might be more harmful than useful. The issue was never completely resolved, however, because the older endarterectomy studies contained a number of flaws. Yet after another pair of multicenter randomized clinical trials was undertaken, one in North America[21] and one in Europe,[22] in early 1991, many were surprised by the announcement of termination of one arm of each of the studies because of evidence documenting overwhelming clinical benefit.

We can learn much from examination of the North American study. Fifty centers participated in the United States and Canada; criteria for participation were combined rates of stroke and death of less than 6% within 30 days of endarterectomy. The subjects were 659 patients experiencing a hemispheric or retinal TIA or nondisabling stroke within the preceding 120 days who were found to have 70 to 99% stenosis of the ipsilateral carotid artery. The study, then, consisted of institutions and surgeons with very low complication rates matched with patients who had major symptoms of severe carotid occlusive disease. The findings will tell us nothing about the value of intervention in asymptomatic patients or in patients with remote or disabling stroke or major organ failure.

The subjects were randomized to either optimal medical therapy alone or optimal medical therapy and carotid endarterectomy. The surgical group had a 65% reduction in the incidence of ipsilateral stroke—a rate of 9% in the surgery group *vs* 26% in the medical therapy alone group over the more than 30 months of follow-up. For major and fatal stroke the figures were 13.1% and 2.5% in the medical and medical plus surgical groups, respectively—an absolute stroke reduction of 10.6% and a relative reduction in stroke of 81%. Death rates were approximately 5% in each group, without statistically significant differences. Major stroke or death occurred perioperatively in 1.2% of the surgical patients. Although medical therapy alone appeared to be more beneficial within the first month because of perioperative complications, by three months surgery showed a clear statistical benefit, which continued to 30 months.

How to apply these findings in clinical practice depends on several considerations. Most important in this study is the 1% rate of combined perioperative mortality and serious morbidity. If the surgical group has a perioperative stroke rate or mortality in the range of 10 to 15%, which is not unheard of, much of the benefit will be lost. The importance of low perioperative mortality and morbidity for the surgical team cannot be overemphasized. Of note is that the incidence of bad events was the same regardless of the severity of baseline risk. That means the risk reduction benefit provided by surgery will be greatest in patients at greatest risk. And even in the high-risk group, gradations occur; medically treated patients with 70 to 90% stenosis had a lower stroke rate than those patients with stenosis of 90 to 99%. In patients with stenosis of less than 70%, the difference between medical and surgical therapy was not dramatic, and that arm of the study is continuing.

The European version of the North American trial reported similar outcomes after three years of follow-up. Surgery provided no benefit and resulted in a statistically greater incidence of stroke or death for the 374 patients with mild, 0% to 29%, stenosis, because strokes were rare with medical therapy in this group. The group with moderate stenosis also did not show benefit, but randomization is continuing and this arm of the study is still in progress because there was no clear difference between surgery with medical therapy and medical therapy alone. Unlike those in the North American trial, participating centers did not have to meet any entry criteria for perioperative complications, and such complications were more common. Although surgical patients with severe stenosis had a 7.5% incidence of perioperative stroke or death and an additional 2.8% incidence during follow-up, medically treated patients had an even higher incidence of stroke and death: 16.8% over the three years. The benefit in the surgical group is still strikingly statistically significant, though the benefits have been proportionally diminished from five-fold in the North American trial to less than two-fold because of the perioperative complication rate.

Delaying and perhaps preventing stroke in older persons by medical treatment of hypertension (especially ISH) and nonvalvular atrial fibrillation, and by carotid endarterectomy in those with severe stenosis, is now a highly desirable and achievable goal. Active life span is increased, the health care dollar is stretched to meet additional needs, and life quality is enhanced for older adults.

REFERENCES

1. Barnett HJM, Hachinski VC: Cerebral ischemia: Treatment and prevention. *Neurol Clin* 1992; 10:1-292
2. Garraway WM, Whisnant J: The changing pattern of hypertension and the declining incidence of stroke. *JAMA* 1987; 258:214-17.
3. Baum HM, Manton KG: National trends in stroke-related mortality: A comparison of multiple cause mortality data with survey and other health data. *Gerontologist* 1987; 27:293-300.
4. Whisnant JP, Mellon LJ, Davis PH, et al: Comparison of case ascertainment by medical record linkage and cohort follow-up to determine incidence rates for transient ischemic attacks and stroke. *J Clin Epidemiol* 1990; 43:791-7.
5. Department of Health and Human Services. *Aginq America* 1991. DHHS Publ. No. 91-28001 (FCoA).
6. Probstfield JL, Furberg CD: Systolic hypertension in the elderly: Controlled or uncontrolled. In: Frohlich ED, ed. *Preventive Aspects of Coronary Heart Disease*. Philadelphia: FA Davis Co. Publishers; 1990:65-84.
7. SHEP Cooperative Research Group: Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. *JAMA* 1991; 265:3255-64.
8. SOLVD Investigators: Effect of enalapril on mortality and the development of heart failure in asymptomatic patients with reduced left ventricular ejection fractions. *N Engl J Med* 1992; 327: 685-91.
9. Kannel WB, Abbott RD, Savage DD, et al: Epidemiologic features of chronic atrial fibrillation: The Framingham study. *N Engl J Med* 1982; 306:1018-22.
10. Wolf PA, Dawber TR, Thomas HE Jr, et al: Epidemiologic assessment of atrial fibrillation and risk of stroke: The Framingham study. *Neurology* 1978; 28:973-7.
11. Albers GW, Sherman DG, Gress DR, et al: Stroke prevention in nonvalvular atrial fibrillation: A review of prospective randomized trials. *Ann Neurol* 1991; 30:511-18.
12. Stroke Prevention in Atrial Fibrillation Investigators: Stroke prevention in atrial fibrillation study: Final results. *Circulation* 1991; 84:527-539.
13. The Boston Area Anticoagulation Trial for Atrial Fibrillation Investigators: The effect of low-dose warfarin on the risk of stroke in patients with nonrheumatic atrial fibrillation. *N Engl J Med* 1990; 323:1505-11.

YOCON®
YOHIMBINE HCI

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of YOCON® 1/12 gr. 5.4mg in bottles of 100's NDC 53159-001-01, 1000's NDC 53159-001-10 and Blister-Paks of 30's NDC 53159-001-30

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85

Available at pharmacies nationwide
PALISADES
PHARMACEUTICALS, INC.
219 County Road
Tenafly, New Jersey 07670
(201) 569-8502
(800) 237-9083

14. Petersen P, Godtfredsen J, Boysen G, et al: Placebo-controlled, randomized trial of warfarin and aspirin for prevention of thromboembolic complications in chronic atrial fibrillation: The Copenhagen AFASAK study. *Lancet* 1989; 1:175-9.
15. Ezekowitz MD, et al: Warfarin in the prevention of stroke associated with nonrheumatic atrial fibrillation. *N Engl J Med* 1992; 327: 1406-12.
16. SPAC: Predictors of thromboembolism in atrial fibrillation: I. Clinical features of patients at risk. *Ann Intern Med* 1992; 116:1-5.
17. SPAC: Predictors...: II. Echocardiographic features of patients at risk. *Ann Intern Med* 1992; 116:6-12.
18. Antiplatelet Trialists' Collaboration: Secondary prevention of vascular disease by prolonged antiplatelet treatment. *BMJ* 1988; 296:320-31.
19. The Dutch TIA Trial Study Group: A comparison of two doses of aspirin (30 mg *vs* 283 mg A day) in patients after a transient ischemic attack or minor ischemic stroke. *New Engl J Med* 1991; 325:1261-6.
20. SALT Collaborative Group: Swedish aspirin low-dose trial (SALT) of 75 mg aspirin as secondary prophylaxis after cerebrovascular ischaemic events. *Lancet* 1991; 338:1345-9.
21. North American Symptomatic Carotid Endarterectomy Trial Collaborators: Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 1991; 325:445-53.
22. European Carotid Surgery Trialists' Collaborative Group: MRC European carotid surgery trial: Interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis. *Lancet* 1991; 337:1235-43.

CALL FOR PAPERS

Members of the Connecticut State Medical Society reading papers before other organizations are requested to submit their papers to the JOURNAL for consideration by the Board of Editors for publication. Authors preparing manuscripts for submission to *Connecticut Medicine* should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office (also printed in *Connecticut Medccine*). Adherence to the instructions will prevent delays both in acceptance and in publication. A copy of the manuscript on floppy disk (3-1/2" or 5-1/4") should be submitted along with the hardcopy.

Please send them to:

Robert U. Massey, M.D.
Connecticut Medicine
160 St. Ronan Street
New Haven, CT 06511

Congestive Heart Failure in the Elderly

JAMES E.C. WALKER, M.D.

Abstract—Changes occurring in the cardiovascular system as healthy subjects age, specifically vascular rigidity and decreased ventricular compliance, increase risks for the development of systolic hypertension, left ventricular hypertrophy, and diastolic dysfunction. These changes of "usual aging" act as independent risk factors which increase the frequency of morbid events accompanying hypertension and coronary artery disease as these diseases increase with age. The rate of congestive heart failure, for example, increases exponentially, developing a three-year mortality of 80% in patients older than 65.

Advances in prevention, diagnosis, and management over the past decade have delayed the onset of cardiovascular events, extending healthy life span remarkably. These advances are reviewed, placing special attention on the changes of aging that effect the clinical management of older patients.

Introduction

THE annual incidence of congestive heart failure (CHF) increases exponentially with age, from a rate of two per 1,000 in the fifth decade of life to 50 per 1,000 in the eighth decade; this increase is accompanied by a three-year mortality rate of 80% for those older than 65.[1] These ominous data accurately reflect the morbid nature of the diseases that cause CHF, hypertension (HTN), and coronary artery heart disease (CAD), and also reflect the often unappreciated fact that aging is, of itself, an independent risk. Age joins the better-known risk factors of smoking, diabetes, lipidemia, HTN, and ventricular hypertrophy to increase independently the probability of a cardiovascular event (stroke, heart attack, and CHF).[2] As changes in life style and medical advances have reduced the impact of these risk factors and delayed cardiovascular events to later ages, studies of the separate effects of aging on the cardiovascular system of disease-free elders have been productive.[3] This paper reviews the cardiovascular changes that accompany aging, indicating how they alter the risks, presentations, and management of CHF in the older patient.

Cardiovascular Changes of Usual Aging

Longitudinal studies of humans carefully screened to eliminate cardiovascular disease,[3] and of disease-free animals,[4] demonstrate aging changes in cardiac afterload, diastolic filling, and systolic function. These changes represent the cardiovascular risk factors attributable to aging of most populations, and have been termed "usual aging"; they need to be recognized and their dynamics understood if success in early diagnosis and management is to be achieved with older patients. While these changes occur in most studied elder subjects, they do not occur in all. Studies of subjects who age free of these changes, "successful aging,"[5] hold prospects of future challenge for prevention.

A. *Increased afterload.*—The decrease in peripheral vascular compliance associated with age-related increase in arterial rigidity has long been recognized, and distinguished from the atherosclerotic plaques of vascular disease; only recently have the long-term effects of decreased compliance on increasing cardiac afterload been appreciated. As peripheral vessels lose their elastic recoil, and the aortic root dilates, the ventricles meet a large inertial load in early systole and must assume a larger role in starting and maintaining forward flow through sys-

---

JAMES E.C. WALKER, M.D., Professor of Medicine, Emeritus, University of Connecticut School of Medicine, Farmington; Associate Director, Travelers Center on Aging, University of Connecticut Health Center, Farmington.

Acknowledgements: The author is grateful to Mrs. Kathleen Locher for manuscritpt preparation, and to Drs. Richard W. Besdine and James O. Judge for manuscript review and suggestions.

tole.[4] This increase in peripheral vascular impedance is at least in part responsible for the age-related increase in systolic pressure (beginning in the fifth decade), and probably plays a role in the enlargement of the left ventricle noted in aging.[6] Both left ventricular hypertrophy (LVH) and isolated systolic hypertension (ISH) are independent risk factors for cardiovascular events and CHF in particular.[2,6]

B. *Systolic function.*—The aging heart has diminished cardioacceleration (chronotropic) response to adrenergic stimuli. Cardiac output remains normal with moderate activity in aging because stroke volume increases to maintain cardiac output in the face of slower rates.[4] Elongation of the myofibril and increase in end-diastolic volume (the Frank-Starling mechanism) produce greater stroke volume. In addition to the chronotropic response, the aging heart demonstrates a diminished inotropic response to maximum exercise. The force of systolic contraction is unchanged, the duration of contraction is prolonged, and end-systolic volume is higher in elderly subjects under strenuous activity. This occurs to a much lesser degree in the older athlete.

C. *Decreased diastolic filling.*—The rate of diastolic filling decreases 50% by the seventh decade in the clinically well elderly[7] and in disease-free senescent rats.[4] Slowed relaxation of the ventricle in early diastole, when it relaxes most rapidly (the isovolumic phase), is most responsible; ventricular filling becomes more dependent on atrial contraction. The characteristic echocardiographic evidence of these changes, slower mitral valve flow rate and later ventricular contraction, are seen in well elderly, but also in young patients with LVH due to HTN or aortic stenosis. The diastolic filling deficit cannot be attributed solely to the increased afterload seen in elderly subjects because it also occurs in their right ventricles which have no increased afterload.[6] These changes must be an effect of aging common to the cardiac myocyte of both ventricles.

The pioneering work of Lakatta and colleagues demonstrating the slower departure rate (efflux) of calcium from the cytoplasm of the cardiac myocyte (the cytosol) during diastole in healthy old rats[4,6] provides a linkage between usual aging and cardiac disease.[7-11] The rate of calcium efflux from the cytosol during diastole determines the speed of ventricular relaxation (lusitophy), and is dependent on ATP as the energy source. The decrease in the diastolic filling rate seen with usual aging, and with LVH at any age, also occurs in the diastolic failure associated with CAD and HTN, in which there is a similar delay in clearing cytosolic calcium from the cytosol.[8-10]

D. *Effect of exercise.*—It is interesting that the increased diastolic and stroke volume used by the aging heart to maintain cardiac output at slower heart rates is similar to the physiologic (eccentric) hypertrophy used by the athlete's heart to improve its output efficiency under stress of rapid heart rate.[8,11,12] Unlike the concentric hypertrophy occurring with the increased afterload of aging or disease, which achieves its mechanical advantage by increasing the diameter of the ventricle wall relative to volume, eccentric hypertrophy increases diastolic volume relative to ventricular wall diameter to increase stroke volume. Physiologic hypertrophy enables the athlete to keep a lower heart rate with exertion, preserving time for diastolic filling. The increased end-diastolic volume of the older heart enables it to maintain cardiac output under the restraint of limited increases in heart rate. Interestingly, endurance training of older men enhances their stroke volume by physiologic hypertrophy, a mechanism very similar to that used by their hearts during aging, increasing their systolic performance up to the level of young sedentary men.[13] Recent studies have demonstrated that diastolic filling rates may also be improved by exercise training. Training, known to increase diastolic filling in younger subjects, has been shown to have similar effects in older subjects; filling rate increases are equal to those of younger sedentary controls.[14]

The role of physical activity, both in improving diastolic filling rates and in enhancing systolic function, is intriguing and has important application to modifying some of the usual cardiovascular changes of aging.

Congestive Heart Failure in the Elderly

Recognition of the special problems brought by aging to elder patients with CHF has increased rapidly during the past decade.[3,8-11] The development of decreased diastolic filling rates[15,16] and likelihood of LVH,[4,17-19] increases the risk of diastolic failure with age. Because the prime cause of systolic failure (CAD) also increases with age, and the management of systolic and diastolic CHF are very different, it is important to distinguish between the two.

Systolic failure, a failure of output marked by incomplete ventricular emptying (low ejection fraction), is most commonly due to the ischemia of inadequate coronary blood supply. This ischemia of supply (supply ischemia) is to be distinguished from the ischemia of demand (demand ischemia) occurring in the hypertrophied heart when the work load overcomes a supply which may be diminished by microvascular disease.[18] Decreased cardiac output activates the increased renin, angiotensin, and aldosterone cascade, which maintains blood pressure and organ blood supply by strategic vasoconstriction and dilation. Recent reports suggest that when CHF is accom-

UNIVERSITY OF MARYLAND BALTIMORE

panied by myocardial damage, angiotensin messenger RNA stimulates myocardial modeling.[8] Atrial naturetic peptide (ANP) acts as a diuretic in these events to modify (not very effectively) the salt and water overload that may result.[3]

Diastolic failure is a filling defect, not an output defect. If the ejection fraction is normal, and especially in the absence of coronary insufficiency, the likelihood of CHF due to diastolic failure is high.[8,9] The mark of diastolic failure is high ventricular diastolic filling pressure, indicating less ventricular compliance (stiffness), and an enlarged atria with delayed emptying. The elevated left and right atrial pressures are reflected by pulmonary congestion and venous distension, respectively. The presence of LVH, whether due to the increased afterload of valvular disease or the peripheral vascular resistance of HTN, signals the likelihood of diastolic dysfunction.[8,16,17,20] Systolic failure is less likely unless there is coincident coronary disease sufficient to create demand ischemia.

The LVH of increased afterload (concentric hypertrophy) is to compensate for an increased workload and is the companion of diastolic failure; the physiologic (eccentric) hypertrophy of the athlete and its relative, the Starling mechanism seen in the aging heart, is to maintain output at lower heart rates.[6,16-19] Unlike concentric hypertrophy, which thickens the myocardial wall to gain mechanical advantage over a given ventricular volume, eccentric hypertrophy stretches the myocardial fiber and increases ventricular volume. Stroke volume and diastolic filling are increased by eccentric hypertrophy, while concentric hypertrophy jeopardizes cardiac output by decreasing diastolic filling rates. The development of concentric LVH, occurring with the afterload increases of age and HTN, counteracts the development of the Starling-driven increase in end-diastolic volume that maintains stroke volume. The importance of preventing LVH in the elderly becomes all the more apparent in this scenario.

Diagnosis

The presenting symptoms of CHF in the elderly are often less organ-specific than in the young.[3,7,9] Generalized fatigued or confusion may precede exertional dyspnea in the elderly, and edema has many causes other than CHF. In the physical examination, the finding of rales may not be specific for CHF, but elevated jugular venous pulse or hepatojugular reflux are excellent confirmatory signs. Cardiac murmurs indicative of valvular disease, arrhythmia, and cardiomegaly, especially in the presence of the tapping apical impulse of LVH, are important findings in the assessment of CHF in both young and old. The presence of a fourth heart sound is common in the elderly as diastolic filling becomes more dependent on auricular contraction, but an early diastolic gallop (S3) is an indicator of pathology and correlates well with the increased LV filling pressure of diastolic dysfunction.

It is especially important in the elderly to identify predisposing, often preventable, factors of CHF early, and to distinguish diastolic from systolic failure; laboratory studies developed in the 1980s permit easy differentiation. Left ventricular hypertrophy, an independent risk factor for morbidity and mortality, and closely associated with diastolic dysfunction, is difficult to identify early by physical examination, chest roentgenography, and EKG; echocardiogram (ECHO) measures ventricular wall thickness and cavity size and distinguishes concentric from eccentric hypertrophy.[20] Valvular disease, diastolic filling rate, and systolic ejection fraction can also be determined by ECHO, making it an essential procedure to assess etiology and management of cardiac failure.[16,21] The presence of normal systolic function in the face of slow diastolic filling and jugular venous distention, for example, indicates diastolic rather than systolic failure.

Two recently developed, noninvasive procedures add objective data to clinical assessment of CHF, enabling early diagnosis and treatment, shown to reduce mortality and hospitalization rates for CHF. A noninvasive method of determining pulmonary capillary wedge pressure (PCWP) has been developed, based on the response of a pressurized finger arterial pulse wave detector to the Valsalva maneuver.[22] PCWP indicates left atrial pressure and, as it becomes more widely available, can assist in the early diagnosis and clinical management of CHF.

Increased levels of atrial naturetic peptide (ANP) are an early warning sign of CHF.[23] ANP, elaborated by granules in atrial myocytes when they are stretched by increased volume or disturbed by atrial fibrillation, acts as an endogenous digitalis-like inotrope and stimulates diuresis.[24] ANP levels, determined by laboratory testing, available to most hospitals, increases independently with disease-free aging, with hypertension, and with venous volume increases.[25] In a prospective study of nursing home patients, a past history of CHF and an ANP of greater than 200 predicted development of CHF within a year with an accuracy of 68%. Elevated ANP helps detect CHF early in the elderly, especially in patients with a positive past history.

Therapy

The effects of aging on the cardiovascular system, including decreases in peripheral vascular and ventricular compliance, weaken homeostatic responses of the elderly, making them more prone to cardiovascular disease and to side effects of medication. The clinically-well elderly face the onset of cardiovascular disease with a higher afterload, a tendency to LVH, a delayed filling rate of both the left and right ventricles, a lessened inotropic and chronotropic response to stress, and a diminished orthostatic response. The recognition of these changes in the elderly enables the clinician to select CHF medications that maximize benefit while anticipating likely side effects. With these points in mind, therapy of CHF in the elder patient, as in the younger patient, is directed to the reduction of preload and afterload and to systolic/diastolic dysfunction accordingly.

Preload reduction.—Diuretics and vasodilators reduce arterial and venous pressure, and remain a hallmark of therapy for CHF in the elderly, but their side effects are more common and troublesome.[3,9] Thiazide and loop diuretics are effective for volume overload; if the tendency of the elderly to volume and sodium depletion is monitored, they are safe. Potassium depletion is less frequent due to decreased glomerular filtration rate (GFR) in the aging kidney. Since causes for ankle edema increase with age, it is important to verify the presence of volume overload by history and other physical findings, such as neck vein distension or pulmonary rales, before using diuretics. Periodic assessment of electrolyte and volume status is valuable.

The selection of venous rather than arterial vasodilators hinges on identifying the systolic and diastolic components of CHF. As diastolic filling rates decrease with age, and the prevalence of diastolic failure increases, the proper use of diuretics and nitrates in the reduction of venous pressure assumes more importance. A reduction in venous pressure, whether produced by the volume depletion of diuretics or the venous dilatation of nitrates, will decrease diastolic pressure and diminish the edema and dyspnea of CHF; over-treatment will reduce diastolic filling, cardiac output, and organ perfusion. Lessened filling pressure in the ventricle during diastole improves coronary blood flow on the one hand, but can also decrease systolic output on the other. Orthostatic hypotension, a common problem often due to reduced baroreflex response or autonomic failure in the elderly, needs to be monitored as a potential early side effect. The capacity to assess PCWP by noninvasive means,[22] provides a more sensitive measure of therapeutic effect than rales or neck vein distention; following postural blood pressures can minimize the risks of hypotension.

Afterload reduction.—Reduction of afterload is accomplished to some degree by nitrates and diuresis, but more effectively by arterial vasodilation. Although hydralazine and isosorbide dinitrate are still used successfully in the treatment of CHF,[26] the sympatholytic vasodilators of the past have been mostly replaced by calcium channel blockers and by angiotensin converting enzyme (ACE) inhibitors. ACE inhibitors (ACEI) have added a separate mechanism of reducing LVH, to the benefits of lowering arterial pressure, and with it, the opportunity to prevent, or at least delay, the onset of CHF. Longitudinal studies reported in 1992 demonstrate a 37% reduction in severe CHF and a 20% reduction in mortality for asymptomatic patients receiving captopril or enalapril within 16 days of myocardial infarction.[27,28] These benefits increased with age and were greater with increased disease severity.

Calcium channel blockers, while not sharing the ACEI benefit of a separate mechanism to reduce LVH, reduce afterload effectively by causing peripheral vasodilation (the nifedipine group). Those with negative inotropic effects (verapamil and diltiazem group) are contraindicated in systolic failure, but may be very useful in diastolic failure (see below).[29]

Diastolic failure.—Because of the increased likelihood of diastolic dysfunction with age, and the detrimental effects that medications commonly used for systolic failure may have on diastolic function, it is especially important in the elderly to distinguish diastolic from systolic failure. Inotropic agents, such as digitalis, which enhance contraction by reducing the diastolic efflux of calcium from the myocyte, can increase ventricular stiffness, and reduce diastolic filling, thereby compounding diastolic failure.[3,9] Diastolic filling can also be jeopardized by too enthusiastic diuresis or use of nitrates (reducing diastolic pressure), and by decreasing the filling time (tachycardia).

In the absence of coronary insufficiency, and in the presence of LVH with or without HTN, the likelihood of CHF due primarily to diastolic failure is high in the elderly. Digitalis is contraindicated except to control rate of atrial fibrillation. Diuresis and preload reduction to control dyspnea and edema, while maintaining filling pressure, is first priority. A calcium channel blocker, such as verapamil, which decreases calcium myocyte entry and therefore the load required to clear during diastole, is then indicated to improve filling rate. Verapamil may also exert its salutary effect on diastole by reducing LVH; its use does not impair systolic function.[29]

Systolic failure.—Therapy of systolic failure must address both the cause of the failure (supply or demand ischemia) and the result (systolic dysfunction). Elderly patients with acute myocardial infarct (AMI), including those 80 and older, are excellent candidates for thrombolytic therapy and for angioplasty. Following AMI, thrombolytic therapy reduces one month mortality rates from 37% to 20%; the older the age the larger the benefit.[30] Angioplasty demonstrates equal benefits, with a lower rate of restenosis than thrombolysis and greater benefits to higher risk groups, including those 75 years and older.[31]

Demand ischemia results in systolic dysfunction as the available blood supply is exceeded by an increasing cardiac workload. These events commonly occur in older patients with LVH, often accompanied by diastolic dysfunction; microvascular coronary disease progresses or afterload increases to produce relative insufficiency.[3] The best treatment is to reduce afterload, and thereby reduce workload, and to improve coronary circulation. Nitrates, especially isordil, and the afterload reducing agents reviewed above, are useful. The two-fold importance of reducing LVH should be recognized: 1). the workload demand on coronary flow is decreased as ventricle mass is reduced; and 2) diastolic function, especially filling rate, is improved.

Treatment of systolic dysfunction in the elderly CHF patient has been modified as the effects of inotropic agents, especially the glycosides, on cytosolic calcium have become more fully understood.[3,8,10,11] Increasing the force of contraction by increasing the systolic level of cytosolic calcium, the mode of action of digoxin, increases the load of calcium to be cleared in diastole prolonging diastolic filling time. Using a cardioselective beta blocker, such as metoprolol, to up-regulate beta receptors, increasing cAMP to improve contraction and perhaps relaxation, is an attractive and promising alternative.[32] Because diastolic dysfunction is increasingly common with aging, the use of cardiac glycosides, except in dilated myocardiopathy or for rate control of atrial fibrillation, is rarely advisable in older patients. Use of other inotropes, including the phosphodiesterase inhibitors, may increase rather than reduce mortality in elder patients.[3]

Prevention

Attention to the well-recognized CHF risk factors of HTN and CAD is a good investment in the elderly. Treatment of isolated systolic hypertension (ISH) in the elderly reduced the five-year rate of CHF events by more than one-half and the rate of AMI by nearly one-third.[34] Studies of other cardiovascular risk factors in the elderly, including smoking, inactivity, and probably LVH, cholesterol, and obesity, indicate similarly rewarding potential, even when initiated in later life.[34] The evidence that physical activity can modulate the age-related decline in both systolic contraction and diastolic filling in the elderly is especially relevant to the older persons who abandon exercise upon retirement.[13,14]

The age-associated increase in LVH, probably related to gradual increases in afterload, represents an additional risk factor that usual aging adds to cardiovascular events.[19] The risks associated with LVH caused by HTN or aortic valve disease are decreased by reducing afterload; so too are the attendant problems of diastolic dysfunction and demand ischemia ameliorated. The mortality and risks of CHF associated with the remodeling (hypertrophy) following an AMI are decreased by ACE inhibitors.[27,28] Although the LVH of aging which results from the increased afterload of vascular rigidity, is theoretically not amenable to vasodilator or diuretic therapy, no prospective studies have specifically addressed LVH in the elderly.

Risk reduction in the elderly has the great attraction of delaying disability and extending active life span. The economic and quality of life advantages are compelling. The success in delaying CAD by modifying health behavior, and the benefits of treating HTN, validate prevention, "even in later years." Preventing or delaying the onset of CHF by these means, and by the use of ACEI following AMI, are further opportunities for success.

A more fundamental understanding of the changes of "usual aging" will be required to reduce risks to the cardiovascular system (primary prevention).[5] A more clear understanding of how the successes in "successful aging" are achieved, would be a wonderful advance to increasing the healthy life span.

REFERENCES

1. Kannel WB: Epidemiologic aspects of heart failure. *Cardiol Clin* 1989; 7:1.
2. Kannel WB: Epidemiology of cardiovascular diseases in the elderly. *Cardiol Clin* 1992; 22(2):9-22.
3. Luchi RJ, Taffet GE, Teasdale TA: Congestive heart failure in the elderly. *J Am Geriatr Soc* 1991; 39:810-25.
4. Lakatta EG, Mitchell JH, Pomerance A, et al: Human aging: Changes in structure and function-cardiovascular disorders in the elderly. *J Am Coll Cardiol* 1987; 10:42A-47A.
5. Rowe JW, Kahn RL: Human aging: Usual and successful. *Science* 1987; 237:143-4.
6. Lakatta EG: Do hypertension and aging have similar effects on the myocardium? *Circulation* 1987; 75:69-77.
7. Wei JY: Age and the cardiovascular system. *N Engl J Med* 1992; 327:1735-9.
8. Grossman W: Diastolic dysfunction in congestive failure. *N Engl J Med* 1991; 325:1557-64.
9. Bonow RO, Udelson JE: Left ventricular diastolic dysfunction as a cause of congestive heart failure. *Ann Intern Med* 1992; 117:502-10.
10. Morgan JA: Abnormal intercellular modification of calcium as a major cause of cardiac contractile dysfunction. *N Engl J Med* 1991; 25:625-32.
11. Katz AM: Cardiomyopathy of overload. *N Engl J Med* 1990: 322:100-10.
12. Lakatta EG: Starling's law of the heart: Interaction of muscle length and calcium activation. *J Am Coll Cardiol* 1987; 10: 1175-64.
13. Ehsani AH, Ogawa T, Miller TR, et al: Exercise training improves left ventricular systolic function in older men. *Circulation* 1991; 83:96-103.

14. Forman DE, Manning WJ, Hauser R, et al: Enhanced left ventricular diastolic filling with endurance training. *J Gerontol* 1992; 47:M56-M58.

15. Arora RR, Macha J, Goldman ME, et al: Atrial kinetics and left ventricular diastolic filling in healthy elderly. *J Am Coll Cardiol* 1987; 9:1255-60.

16. Cacciapuoti F, D'Avino M, Lama D, et al: Progressive impairment of left ventricular diastolic filling with age. *J Am Geriatr Soc* 1992; 40:245-50.

17. Topol EJ, Traill TT, Fortuin NJ, et al: Hypertensive hypertrophic cardiomyopathy of the elderly. *N Engl J Med* 1985; 312:277-83.

18. Frohlich ED, Apstein C, Chobanian AV, et al: The heart in hypertension. *N Engl J Med* 1992; 327:988-1008.

19. Aronow WS: Left ventricular hypertrophy. *J Am Geriatr Soc* 1992; 40:7180.

20. Levy D, Anderson KM, Savage DD, et al: Echocardiographically detected left ventricular hypertrophy: Prevalence and risk factors. *Ann Intern Med* 1988; 108:7-13.

21. Levy D, Garrison RJ, Savage DD, et al: Left ventricular mass and incidence of coronary heart disease in an elderly cohort. *Ann Intern Med* 1989; 110(2):101-7.

22. McIntyre KM, Vita JA: A non-invasive method of predicting pulmonary capillary wedge pressure. *N Engl J Med* 1992; 327:1715-20.

23. Davis KM, Fish LI: Atrial naturetic peptide levels in the prediction of congestive heart failure risk in frail elderly. *JAMA* 1992; 267:2625-29.

24. Haddy FJ: Endogenous digitalis-like factor. *N Engl J Med* 1987; 316:621-2.

25. Ezaki H, Matuschita S, Shiraki M, et al: Clinical evaluation of plasma levels of atrial naturetic peptide in elderly patients with heart disease. *J Am Geriatr Soc* 1988; 36:537-41.

26. Cohn JN: Prevention of heart failure—a new agenda. Editorial. *N Engl J Med* 1992; 327:725-7.

27. The SAVE Investigators. Effects of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction. *N Engl J Med* 1992; 327:669-77.

28. The SOLVD Investigators. Effects of enalapril on mortality and the development of heart failure in asymptomatic patients with reduced left ventricular ejection functions. *N Engl J Med* 1992; 327:685-91.

29. Schulman SP, Weiss JL, Becker LC, et al: Effects of antihypertension therapy on left ventricular mass in elderly patients. *N Engl J Med* 1990; 32:1350-56.

30. Krumholz HM, Pasternak RC, Weinstein MC, et al: Cost effectiveness of thrombolytic therapy with streptokinase in elderly patients with suspected myocardial infarction. *N Engl J Med* 1992; 327:7-13.

31. Grines CL, Browne KF, Marco J, et al: A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. *N Engl J Med* 1993; 328:673-9.

32. Eichhorn EJ: The paradox of beta-adrenergic blockade for management of congestive heart failure. *Ann Intern Med* 1992; 92:527-38.

33. SHEP: Cooperative Research Group. Prevention of stroke by antihypertensive treatment in older persons with isolated systolic hypertension. *JAMA* 1991; 285:3255-64.

34. Paffenbarger RS Jr, Hyde RT, Wing, et al: The association of changes in physical-activity and life style with mortality among men. N Engl J Med 1993; 328:538-45.

UNIVERSITY OF MARYLAND AT BALTIMORE

Urinary Incontinence for the Primary Care Physician

FITZHUGH C. PANNILL, III, M.D.

ABSTRACT—This article reviews the recent data on the etiology, evaluation, and treatment of urinary incontinence in older adults. While urinary incontinence remains a major problem for older people, many of these patients never receive medical evaluation or treatment. The nonspecialist can make substantial inroads into incontinence by identifying affected patients, treating medical causes of incontinence effectively, and evaluating bladder and urethral function using simple techniques. While many patients may need referral, the cause of incontinence in most older adults can be identified and effectively treated by nonspecialists. Behavioral methods are becoming increasingly important in this effort. In office practice, up to 80% of all patients will respond with decreases in incontinence of at least 50%. Controlled studies indicate between one third and one half of nursing home patients will be equally improved. Consequently, there is good reason for substantial optimism with this problem.

Introduction

URINARY incontinence (UI) is frequently a severe problem for older people in both outpatient and inpatient practice.[1,2,3] Despite the fact that UI causes substantial social, economic, and medical difficulties, and at least half of the patients can be improved substantially without extensive evaluations, many incontinent patients never come to medical attention.[4,5] The following discussion will outline a practical approach to incontinence, designed for office practice. Excellent, extensive reviews are available that provide more information,[2,4,5,6] including a recently published review of diagnosis and treatment guidelines prepared by national experts for the U.S. Department of Health and Human Services.[7]

Frequency

Surveys of unselected older adults in the community demonstrate that between 8% and 34% are incontinent of urine.[1,7] The most representative studies, focusing on patients similar to those seen in medical offices, show a 30% prevalence of incontinence.[8] UI is at least twice as common in women than men.[8] Forty-five percent of older patients admitted to hospitals are incontinent at some time during their stay; most remain incontinent after discharge.[9] In nursing homes and other institutions, the prevalence rises to 60 or 70%.

There are substantial costs associated with incontinence. UI was estimated to cost the nation $10 billion in 1987, not including indirect costs.[10] This was more than the costs of AIDS at that time. The costs in loss of patient morale, social isolation, immobility, and unnecessary institutionalization are far higher. Up to 70% of incontinent patients in most studies also report altering travel patterns, staying home, or trouble with embarrassment and family.[3]

Physiology and Pathophysiology

The central nervous system (CNS) controls and coordinates the bladder and urethra to maintain appropriate storage and release of urine.[2] The urinary bladder muscle, or detrusor, relaxes slowly without much increase in bladder pressure as urine volume increases. Resting urethral pressure is maintained at levels higher than bladder pressure to prevent urinary leakage. With bladder filling, proprioceptive receptors in the bladder wall transmit an increasing frequency of sensory impulses through the sacral nerves and spinal cord to the detrusor motor nucleus in the pons. To initiate urination at any bladder volume,

---

FITZHUGH C. PANNILL, III, M.D., Assistant Professor of Medicine, Yale University School of Medicine, New Haven and West Haven Veteran's Affairs Medical Center, West Haven.

this pontine nucleus coordinates a discharge of neurons through the spinal cord causing the detrusor to contract; at the same time the urethral sphincter relaxes. A collection of neurons in the frontal lobe can inhibit the pontine center and, consequently, inhibit detrusor contractions as bladder volume increases. This inhibitory center develops after birth, and allows substantial increases in bladder capacity without detrusor contractions. In the adult, voiding occurs at volumes of 300-500 ml when bladder sensation comes to consciousness. Frontal lobe inhibition decreases, allowing detrusor contraction and urethra relaxation. Bladder innervation is provided by the parasympathetic autonomic nervous system (mediated by acetylcholinergic neurons), while the urethra is innervated by the alpha sympathetic system.[2,5] These differences are reflected in the respective effects of different drugs on the bladder and urethra.

Additional components of urethral pressure are important in certain circumstances. The external sphincter (striated muscle) is under conscious control and can prevent urine loss for short periods of time. The internal sphincter (smooth muscle) is not under conscious control, but with tonic contractions can prevent urine leakage at wide ranges of bladder pressure. Estrogen dependent urethral mucosal thickness also provides increased resistance to urine flow in women. Finally, normal pelvic geometry allows the transmission of intraabdominal pressure to the proximal third of the urethra, in addition to the bladder. During activities that increase intraabdominal pressure (coughing, sneezing, etc.), incontinence is prevented because urethral pressure rises concomitantly with bladder pressure. Pelvic muscle laxity in older women decreases this pressure transmission by altering normal pelvic geometry and increases the likelihood of incontinence.[2,5]

Table 1 details the major causes of established incontinence due to bladder pathology.[2,5,6] Urge incontinence, characterized by loss of large volumes of urine and a strong urge to void, occurs when CNS pathology [5,6] prevents the frontal lobe inhibition of the bladder and uninhibited bladder contractions (referred to as "detrusor hyperreflexia" if CNS pathology is present) develop.

Table 1.—Summary of Established Incontinence

Incontinence Type	Etiology	Prevelance*	Symptoms	Signs	Parameters	Voiding Urodynamics	Treatment
Urge	1. Defects in central nervous system inhibition (detrusor instability) 2. Bladder irritation 3. Idiopathic deconditioning	Com: 40% NH: 60%	Urgency, frequency, nocturia, loss of large amounts of urine	Underlying central nervous system disorders Urinary tract infection or vaginitis as irritative lesions	Loss of large urine volumes (> 50 ml) Small bladder capacity with frequent losses	Uninhibited contractions Small bladder capacity Normal postvoid residual	Anti-cholinergic medications Treat source of irritation Biofeedback Habit retraining
Stress	1. Pelvic muscle laxity 2. Neuropathy 3. Urological surgery	Com: 60% (mostly women) NH: 25%	Loss with exercise, coughing	Leakage of small amounts with coughing Atrophic vaginitis	Loss of small urine volumes Daytime incontinence (correlates with exercise)	Normal	Surgery Estrogens (women), alpha-agonists Pelvic strengthening exercises
Overflow	1. Detrusor inadequacy 2. Outlet obstruction 3. Impaired bladder sensation	Com: 10% NH: 15%	Hesitancy, straining, incomplete emptying, frequent small voidings	Palpable bladder Prostate enlargement Difficult catheterization	Nocturia Frequent loss of small (10-50 ml) amounts of urine Poor stream	Poor bladder contractions Bladder capacity > 500 ml Large postvoid residual (> 150 ml)	Surgery Prazosin Intermittant catheterization

*Com indicates living in the community; NH, in the nursing home.

Reprinted with permission from Pannill FC: "Diagnosis and treatment of urinary incontinence," in Stults B, Dere WH, eds, *Practical Care of the Ambulatory Patient.* Philadelphia, Pa: WB Saunders Co., 1988.

Thus, cerebrovascular accidents, Alzheimer's disease, brain tumors, multiple sclerosis, Parkinson's disease, normal pressure hydrocephalus, and spinal cord injury can all cause incontinence in this fashion. Similar symptoms and "detrusor instability" (DI) (uninhibited contractions without CNS pathology) can result when increased bladder sensory impulses from vaginitis, benign prostatic hypertrophy, fecal impactions, or urinary tract infections overcome even normal central inhibitory mechanisms.[2,5,11] Occasionally, patients can develop urge incontinence by repetitive and frequent voiding, shrinking their bladder, and developing irritation as a form of deconditioning.[2]

Stress incontinence (SI) is the second common type of established incontinence.[5,6,12] Ineffective pressure transmission to the urethra due to pelvic muscle laxity results in urine leakage when exercise-induced increases in bladder pressure overcome resting urethral pressure. Damage to the pudendal nerve during childbirth or resulting from chronic constipation is another cause of stress incontinence. Occasionally, urological surgery can also cause sphincter damage. The characteristic sign of stress incontinence is loss of small urine volumes with coughing, exercise, or other pressure "stress." Bladder function is normal.

Overflow incontinence (OI) occurs when bladder contractions can not overcome urethral resistance because of either ineffective detrusor contractions (detrusor inadequacy or atonic bladder) and normal urethral pressure or abnormally high urethral resistance above normal bladder pressure due to an obstruction.[2,5] In the first case, sacral nerve damage results in poor or absent detrusor contractions and large bladder volumes at capacity (over 500cc) and large postvoid residuals. Obstruction at the urethral level, usually due to prostatic hypertrophy, can cause a similar picture, as can neurologically impaired sphincter relaxation. Unlike patients with an atonic bladder, however, patients with obstruction or high urethral pressure will demonstrate normal or increased bladder pressure and occasionally detrusor instability. Residual urine volume in these patients may be normal.[2,5]

There are a number of changes in the bladder and urethra that occur with advancing age that, while insufficient to cause incontinence by themselves, can act in concert with other factors to precipitate incontinence.[2,6] These changes probably explain why urinary symptoms other than incontinence are much more common even in continent elderly than younger patients. Nocturia, in particular, is seen in up to 60% of people over 65.[8] Urethral pressure and bladder capacity have been reported to decrease with aging, and residual urine to increase. Other changes are more significant, as they are similar or even identical to those seen in incontinent persons. For example, 30% of continent men and up to 10% of continent women have some degree of uninhibited detrusor contractions.[2,7] While probably insufficient in themselves to cause incontinence, these contractions can be large enough to cause urinary urgency and force the patients to move quickly to avoid incontinence. Impairment of mobility as with hip arthritis or mild dementia, will precipitate incontinence. These patients require a comprehensive and multifactorial evaluation with therapy aimed at both the immobility and the bladder contractions.[2]

Medical or "transient" causes of incontinence are related to associated diseases, medications, and conditions not directly resulting from bladder pathology.[6] The mnemonic DIAPPERS summarizes these important issues: Delirium (and dementia), Infections (of the urine and vagina), Atrophic vaginitis, Psychological causes (especially depression), Pharmaceutical agents, Endocrine conditions (especially diabetes), Restricted mobility (restraints, environmental barriers, or mobility problems), and Stool impaction.[6] Other causes of polyuria such as congestive heart failure, varicose veins, and cirrhosis should be kept in mind, as reabsorbtion of the dependent edema in these conditions results in increased urine volume at night and nocturnal incontinence.

An additional, important cause of incontinence in older people is "functional incontinence," referring to incontinence that occurs in patients with normal bladder and urethral function.[2] Most of these patients show significant confusion about bathroom locations, immobility, or apraxia due to a variety of physical or cognitive problems. These problems all prevent them from using bathrooms or urinals quickly and effectively.

Little specific data exist to document how frequently most of these problems result in incontinence, and it is difficult to determine with certainty the difference between an associated feature and a causal relationship.[2,4] These problems seem to be more important contributors to incontinence in the institutionalized elderly, where the correction of such "medical" or "transient" causes of incontinence has been shown to be more useful than treatment aimed at the "established" urological causes.[13]

Both acute confusion in delirium, and the chronic apraxia and decline of other self-care skills of dementia cause incontinence. Delirium, marked by a loss of alertness and attention, is usually due to a reversible medical illness such as infection, hypoxia, or drug effect. It is unusual for dementia to cause incontinence in patients who do not show declines in other daily living skills and mental status.[4] Thus UI should not be attributed to Alzheimer's disease in patients who are living independently in the earliest stages of the disease, unless all other causes are excluded.

In most incontinent patients with chronic bacteriuria (up to 30% of all nursing home patients), the bacteriuria is not responsible for the UI.[14] Studies in younger women demonstrate that infections may cause detrusor instability that disappears with treatment of the infection.[15] However, it appears it is unusual for urinary infections to cause UI in older patients without symptoms of dysuria.[14] It is standard practice, however, to eradicate existing bacteriuria before urethral catheterization during the evaluation.[2,5] In our incontinence clinic, we document the effect of this treatment on the frequency of UI and have found a few patients whose UI improves after antibiotic treatment. Atrophy and vaginal irritation also can increase urinary symptoms and UI, especially in women with inflamed, friable vaginal mucosa. As nonovarian estrones are converted peripherally in adipose cells and maintain estrogen sensitive tissues, most postmenopausal women do not develop such vaginitis. Estrogen therapy either vaginally, orally, or transdermally can cure incontinence in some women with urgency symptoms,[16] but treatment is unlikely to be effective unless there are signs of vaginal irritation such as discharge with inflammatory cells and vaginal friability.[13] Fecal impaction seems to cause incontinence by inducing similar bladder irritation.[13] Again, as with UTIs, the effect of therapy for vaginitis or impaction on the incontinence should be clearly documented by repeated assessment of the degree of incontinence immediately after therapy.

Depression severe enough to cause UI is usually severe enough to interfere with many self-care activities and other functions. The endocrine syndromes mentioned usually cause polyuria and UI by overwhelming bladder capacity, although diabetes does cause bladder atonia and overflow incontinence. Any medical problem, or "geriatric syndrome" such as falling, that slows ambulation can also be responsible for UI. Thus, arthritis of the hip and associated muscle atrophy, postural instability, and environmental barriers like stairs may be the cause of UI. If these are discovered, simple measures like a bedside commode may be curative.

Table 2 lists mechanisms and signs and symptoms that suggest either prescription or nonprescription medications may be contributing to incontinence. As data on this subject are scarce, all drugs with potential contribution to UI should be discontinued for a period of time, if possible. For example, polyuria due to diuretics, caffeine, or alcohol frequently causes UI in patients with limited bladder capacity or trouble toileting, but it is difficult to be precise in defining this contribution clinically.[4] Incontinence that

Table 2.—Medications Contributing to Incontinence

Type of Medication	Possible Mechanism	Clues to Diagnosis
Anticholinergics*	Detrusor relaxation, increase residual urine	Hesitancy, straining to void, overflow incontinence, high post-void residual
Diuretics	Increase urine volume	Symptomatic polyuria, frequency, and urgency, incontinence within 2 hours of drug
Sedatives and hypnotics	Sedation, altered mental status	Confusion, delerium, missed bladder clues, inability to toilet, nocturia
Antipsychotics	Sedative and anticholinergic effects; bradykinesia and rigidity	See above Inability to toilet due to immobility
Tricyclic antidepressants and narcotics	Sedative and anticholinergic effects; altered mental status	See above
Alpha-adrenergic blockers†	Decrease urethral resistance	Stress incontinence
Alpha-adrenergic agonists‡	Increase urethral resistance	Hesitancy, straining to void, overflow incontinence, high post-void residual
Calcium-channel blockers	Detrusor relaxation; increased residual urine	Hesitancy, straining to void, overflow incontinence, high post-void residual
Alcohol	Increase urinary volume; sedation, altered mental status	Urgency, frequency, nocturia, sedation, confusion, immobility with inability to toilet

* includes anti-parkinsonian, antihistamines, disopyramide, antispasmodics, antipsychotics, antidepressants
† includes prazosin, methyldopa, reserpine
‡ includes ephedrine, cold remedies, decongestants

Reprinted with permission from Pannill FC: Practical management of urinary incontinence. *Med Clinic NA* 1989; 73:1423-39.

UNIVERSITY OF MARYLAND
BALTIMORE

Table 3.—Clinical Evaluation

History

- Urinary symptoms (frequency, dysuria, straining, decreased stream, incomplete emptying, nocturia)
- Incontinence symptoms (urgency, stress, amount, timing, duration, associations with medications, sleep, exercise)
- Fluid intake and voiding patterns (bedtime drink?, caffeinated beverage)
- Active medical problems: (diabetes, neurological disease, heart failure, G.U. surgery, arthritis, mobility, recent acute illness, pelvic irradiation)
- Medications (prescription, nonprescription)
- Environmental factors (location of bathrooms, ease of access, restraints)
- Incontinence chart

Physical

- Orthostatic blood pressure
- Neurologic (mental status, upper neuron signs, peripheral sensation, motor strength, perineal sensation, sacral reflexes, anal tone)
- Palpable bladder after voiding
- Pelvic examination (vaginitis, atrophy, pelvic floor laxity, prolapse, cystoceles, masses)
- Rectal examination (prostate size, impaction, masses)
- Simple cystometrics

Laboratory

- Urine analysis and culture; measured volumes of urine
- Blood work (electrolytes, glucose, urea nitrogen, and calcium)

Modified from: Pannill FC: "Diagnosis and treatment of urinary incontinence," in Stults B, Dere WH, eds, *Practical Care of the Ambulatory Patient.*, Philadelphia Pa: WB Saunders Co., 1988. Reprinted with permission.

is worse after a morning diuretic also points clearly to a contribution.

The Evaluation of the Incontinent Patient

Table 3 summarizes the areas that should be included in the evaluation of an incontinent patient.[2,4,5,6] In addition to history of the incontinence and associated symptoms, information on self-care activities (dressing, bathing, mobility, feeding, and cooking) and medical problems and medications is essential. Environmental factors and timing of voiding and fluid intake may provide important clues to etiology in patients with functional incontinence. Over the counter medications and caffeinated or alcoholic beverages should not be overlooked as contributing factors. An incontinence chart may provide important clues by documenting frequency of wetting episodes, voiding patterns, and frequency of urination.[5] This can be a simple four-day history of these symptoms that the patient records at two hour intervals while awake. Incontinence charts, repeated after therapy, can reliably document the effect of such therapy, as patients' verbal reports may be unreliable. A number of sample charts are available.[5,17]

The physical examination should include measurement of orthostatic blood pressure, a neurological examination with careful attention to the perineal nerves and central nervous system, and pelvic and rectal examinations. Stress testing and postvoid residual measurements are easily obtained during simple cystometrics. Each patient should also have a urinalysis and culture, and measurements of serum electrolytes, glucose, calcium, and renal function, to exclude metabolic causes of polyuria. A number of clues to etiology provided by the history and physical examination are outlined in Tables 4 and 5.[17]

Cystometrics provide detail about bladder sensation and capacity, contractions, residual volume, and stress incontinence[5,6,18,19] and can be easily performed in the office with simple equipment, as detailed in Table 6.[18,19] A urinalysis is necessary before the procedure to exclude infection. This examination measures bladder capacity, residual urine volume, number and strength of bladder contractions, and presence of stress incontinence (leakage of small urine volume with coughing), or cough induced bladder contractions (leakage of large urine volume). Based on comparisons with more extensive urological evaluations, measurements of bladder capacity and detrusor contractions with simple cystometry have a sensitivity of 75% and a specificity of 79%.[20] Ouslander has defined criteria for referral for formal urological examination, including difficulty with catheterization, a high (greater than 200ml) postvoid residual, and severely limited (less than 100ml) bladder capacity.[18,19] Patients with recent pelvic surgery or irradiation, frequent urinary tract infections, hematuria, marked pelvic prolapse, marked prostatic enlargement, severe hesitancy or interrupted urinary stream, or men with stress incontinence should also be referred, as should patients with stress incontinence who want to consider surgical therapy. Although 50% of the patients in one large study of 168 patients met these criteria for referral, over half needed referral only for stress incontinence or obvious pelvic prolapse.[19] These criteria were useful in identifying the 25 patients (15%) found to have obstruction or atonic bladder by complete urodynamics. Twenty of these 25 (80%) met one of the criteria above. In the other five patients (3% of all 168 patients evaluated but 10% of the 32 men), the obstruction would have been overlooked if the diagnosis has been based on simple cystometrics alone. Men were more likely to have obstruction misdiagnosed than women: 10% (3/32) *vs* 1.4% (2/136). If simple cystometrics alone were used to determine therapy, these patients would have been treated for urge incontinence with potential risk of urinary retention. Using these criteria, women with stress and mixed stress/urge incontinence, patients with normal bladders and likely functional incontinence, and patients of both sexes with urge and no signs of obstruction or atonic bladder will not need referral.

As men are more likely to suffer from obstruction, and simple cystometrics are more likely to miss obstructions in men, male patients need more careful monitoring when treated with anticholinergic therapy for urge incontinence. This monitoring should include repeated measurements of the postvoid residual and serum creatinine, and questioning about symptoms of hesitancy or decreased stream.

Incontinence in Office Practice

Limited data indicate that in women, 60-90% of UI in the community is caused by stress incontinence.[21] This is due to the infrequency of debilitating neurological disease or chronic illness and the generally preserved functional abilities of ambulatory older patients, in contrast to nursing home residents. Consequently, diagnostic strategies are different from those in the nursing home.

In office practice, a positive bladder stress test during simple cystometrics in a female is highly predictive (90% posttest probability) of true stress incontinence and can reliably guide therapy.[22] Detrusor instability accounts for between 20% and 50% of incontinence in ambulatory women and can be reliably excluded if not present on simple cystometrics (8% negative posttest probability).[22] Overflow incontinence, unusual in women, can also be excluded if the postvoid residual is normal.

Table 4.—Clinical Clues to Incontinence by History

Clue	Probable Incontinence Etiology	Diagnostic Usefulness*
Loss of urine only with physical stress	Stress incontinence	Almost diagnostic
Volume: Drops only	Stress	Helpful
Large amount	Not stress	Very helpful
Straining, hesitancy, incomplete emptying	Overflow	Helpful
"Almost make it"	Urge or overflow	Helpful
Only noctural incontinence	Fluid reabsorbtion, bedtime drink, sedative, or alcohol	Helpful
Severe frequency (over 8 times/day)	Small capacity bladder, urge, overflow	Helpful
Incontinence only in morning after medication	Iatrogenic	Very helpful
Dysuria	Acute UTI or vaginitis	Very helpful
Able to hold urine for over four hours consistently	Bladder capacity at least 200 mL	Helpful
Prolonged bed rest—Recent surgery	Overflow	Very helpful
B12 deficiency, diabetes, alcoholism	Underactive detrusor	Helpful
New medication c/w incontinence both temporally & physiologically	Iatrogenic	Very helpful
Recent decline in mobility, mood, mental status, self care abilities	Depression, immobility, or functional incontinence	Very helpful

* These represent estimates for positive predictive values: almost diagnostic (over 90%); very helpful (80-90%) and helpful (70-80%).

Reprinted with permission from Pannill FC: Practical management of urinary incontinence. *Med Clinic NA* 1989; 73:1423-39.

Table 5.—Clinical Clues to Incontinence Etiology and Action Required

Data	Probable Incontinence Etiology	Action
Spasticity, hyperactive reflexes in lower extremities; impaired perineal sensation	Possible spinal cord compression	Immediate neurological referral
Congestive heart failure, venous stasis, fluid retention	Polyuria; fluid reabsorption	Treat underlying problem; reevaluate incontinence
Impaired perineal sensation, absent anal wink, without upper neuron signs	Possible sacral nerve impairment causing noncontractile detrusor	Urodynamics
Central nervous system pathology	Detrusor instability	Urodynamics
Palpable bladder	Overflow incontinence	Urology referral
Marked pelvic prolapse, rectocele, cystocele, pelvic or prostatic mass suspicious for cancer	Pelvic pathology	Refer for gynecological or urological evaluation
Vaginal atrophy, discharge, mucosal irritability	Atrophic vaginitis	Treat, reevaluate incontinence
Fecal mass on rectal	Fecal impaction	Treat, reevaluate incontinence
History of recent or multiple genitourinary surgery or pelvic irradiation	Complex pathology	Refer for evaluation
Markedly large prostate	Prostatic hypertrophy	Refer for evaluation
Hematuria	Bladder or renal pathology	IVP; refer for cystoscopy
Pyuria, bacteriuria greater than 10^5 colonies	Urinary Tract Infection	Treat, reevaluate incontinence
Blood sugar over 200 mg/dL	Diabetic polyuria; high risk detrusor inadequacy	Treat, reevaluate incontinence; urodynamics
Increased calcium or blood urea nitrogen	Polyuria	Treat, reevaluate incontinence

Reprinted with permission from Pannill FC: Practical management of urinary incontinence. *Med Clinic NA* 1989; 73:1423-39.

Men, with a high frequency of BPH induced obstruction (40%) and detrusor instability (30-60%), but only rare stress incontinence, are more likely to have false negative examinations for DI and obstruction on simple cystometrics (negative posttest probability of 10-30%) and need more extensive urological examinations to meet the criteria for referral, or if any suspicion of obstruction or voiding difficulties are present.[22]

Medical, or transient causes, should be identified and treated first in both sexes, with cystometrics to follow if the incontinence persists. Tables 4 and 5 give useful clues to etiology. Patients with referral criteria discussed above need in-depth evaluations by a urologist or gynecologist. Most women, however, will have "simple stress incontinence" with little urgency, no increase in urinary frequency or severe nocturia, a normal examination, and normal PVR.[5] Men are more difficult to characterize and often need more extensive evaluation.

Simple stress incontinence can be treated with good results from surgery under local anesthesia,[23] but many patients may avoid surgery with either pelvic floor exercises[24] or biofeedback.[25] Pelvic floor exercises have also been combined with alpha agonists[24] and estrogens.[7] Between 12% and 20% of patients can be cured and up to 75% improved with exercises alone.[7] Surgery is more effective, with cure rates of 78% to 84%,[7,26] but up to 20% of patients *suffer* side effects.[7] Periurethral injections of collagen offer a promising, effective option that in initial studies appear to cause fewer side effects.[7] FDA approval is pending, however.

To be effective, pelvic floor exercises need to be done properly for a least five months and with 40 to 80 repetitions daily.[24] The length of time and intensity of maintenance treatments is not defined precisely. A number of "do-it-yourself" books are available that can be used by patients with some guidance from medical professionals.[27] Formal biofeedback treatments for stress incontinence, conducted by psychologists, have the potential to cure even the most difficult patients with complex or postsurgical problems.[25] Home biofeedback units are being marketed by commercial interests claiming that patients may benefit from electromyographic stimulation of pelvic muscles as a substitute for exercise programs. The effectiveness has not been studied and the costs are high.

Drugs reported useful for stress incontinence include phenylpropanolamine or alpha agonists (19-60% successful), estrogens (20-89% successful), and both together (37-38% successful).[7,28] Many of these studies suffer from small sample sizes and poorly defined outcome criteria.[2,7] Side effects are usually minor, although estrogens remain under suspicion for causing an increase in uterine and perhaps breast cancer.

Behavioral therapies are also effective for patients with detrusor instability, mixed incontinence (DI and SI), and small capacity bladders without other problems. Bladder training requires a fixed schedule of voiding that is slowly lengthened to increase bladder capacity in combination with specific relaxation exercises designed to prevent incontinence due to detrusor contractions.[29] Several large trials have recently been completed involving patients with both SI and DI. These trials report an overall cure or improvement rate (50% decrease in incontinence) of 70% to 87%.[7,30]

A mild anticholinergic, oxybutinin, remains the mainstay of pharmacological therapy for detrusor instability with improvement reported in between 9% to 56% of patients.[7,31] Less widely studied, but useful in some patients, tricyclic antidepressants (20-77% improvement) are best reserved for healthy patients not at risk for the frequent orthostatic side effects.[7,13] In addition to their modest effect on incontinence, and the relatively small number of studies, the most significant issue with pharmacological therapy remains the incidence of side effects, such as delirium, orthostasis, constipation, and urinary retention. Between 14% and 70% of patients in these studies have dropped out because of the side effects. Low starting doses and slow increments in doses will help minimize these problems.[7,13]

There is increasing evidence that behavioral therapy and exercises may be the treatment of choice for most incontinent patients, with surgery and medications reserved for patients who either meet referral criteria or who fail to improve with behavioral therapy.[7,25] Because behavioral therapy has few side effects, many authorities recommend bladder training before cystometrics;[5,7] a number of patients can be cured without invasive testing at all.

Behavioral therapy and pharmacological intervention can be used together for a synergistic effect. Biofeedback, pelvic floor exercises, and educational training aimed at teaching patients how to control their urinary losses may be combined and have achieved impressive results even in very frail older adults. McDowell et al, were able to reduce incontinence by average of 82% (17 accidents/week to 2.5) in 29 patients using a variety of techniques.[32]

Incontinence in the Nursing Home

Institutionalized patients suffer from a greater frequency of neurological disease and other illnesses, and the relative proportion of detrusor instability and overflow incontinence is much higher than in the community patient. Careful studies by Resnick confirm the predominance of detrusor instability (61%), with atonic bladder (8%), obstruction (4%), and mixed diagnoses (4%) responsible for most of the remainder. Only 21% of the women had stress incontinence.[11]

Given their much higher prevalence of DI, institutionalized patients with uninhibited contractions on cystometrics are more likely to have detrusor instability

Table 6.—Simple Cystometrics

1. Check urinalysis and culture to exclude infection. Treat if present.
2. Patient voids (ideally after usual interval). Volume recorded.
3. Catheterize bladder (sterile technique) with straight 14 FR catheter. Note any difficulty or discomfort of catheterization.
4. Drain and record volume of residual urine. Leave catheter in bladder.
5. Using 60 mL catheter tip syringe as a funnel (plunger removed) slowly fill bladder with sterile water or physiologic saline, 50 mL at a time. Syringe should be no more than 15 cm above symphysis pubis. Record volume.
6. When patient reports urge to void, slow filling to 25 mL at a time. When patient reports "unable to hold anymore," remove catheter. This volume is maximal bladder capacity.
7. If level of fluid in syringe stops falling and rises during filling, the patient is having a bladder contraction, or contracting abdominal muscles. If latter is excluded, and contraction is over 15 mL, this probably represents detrusor instability. Remove catheter after contraction subsides.
8. With catheter out, have patient cough vigorously while standing. Collect any urine leakage on perineal pad and note volume (small or large).
9. Patient voids in commode and volume recorded. Residual volume is the difference between bladder capacity and volume voided.

Adapted from Ouslander[18]

(positive posttest probability of 84%) than patients seen in the office. On the other hand, absence of uninhibited contractions on cystometrics does not reliably exclude DI in institutionalized patients (negative posttest probability of 34%).[22] Stress incontinence may be reliably excluded with a negative stress test in this setting because of its low prevalence (negative posttest probability of 4%).[22] There are few data on the specific criteria for referral in a nursing home. In our practice, most patients can be managed without referral, unless suspicion of cancer exists (hematuria, sterile pyuria, nodular prostate), or the patient is a candidate for surgical relief of an obstruction. Most patients with overflow incontinence, even that due to prostatic hypertrophy, can be safely managed with alpha-blockers and/or intermittent straight catheterization. Finasteride, a prostate alpha reductase inhibitor, shows some promise in shrinking prostatic hypertrophy, but has not been shown to reduce residual urine in these patients.[33]

Oxybutinin and tricyclics can control detrusor contractions[28] but need to be used judiciously, given the incidence of serious side effects such as delirium, orthostasis, and urinary retention.[13] In general, efficacy is limited, and the few well controlled trials in nursing homes have shown little benefit, compared to habit training or placebo.[31] Estrogens have been reported to reduce incontinence by up to 89% in female nursing home patients,[16] but the risk of long-term side effects requires careful documentation of the improvement in individual patients before long-term use is continued.

Behavioral therapies have been particularly well studied recently and are by far the most successful treatment of UI in the nursing home. Hu et al, used hourly checks and prompted voiding in a randomized controlled trial of demented, dependent (24% skilled care; 76% intermediate care) patients who were able to at least recognize their own name.[34] Overall, incontinence was reduced by 26%, with 38% (25/65) of the patients reducing incontinence frequency by at least 50%. More improvement was seen in patients with greater baseline incontinence (more than two episodes per 12 hours), less severe dementia, and more functional dependence. Cystometric diagnosis was not useful in predicting outcome except that patients with normal bladder capacity showed more improvement than those with small bladders. Schnelle studied a similar prompted voiding program in 126 demented patients, and reduced incontinence episodes by 50%.[35] Using individualized habit training, Colling was able to reduce incontinence by at least 25% in one third of the 51 patients studied, while 37 controls showed an increase in incontinence during the same period.[36] These trials provide optimism that incontinence in nursing home patients can be improved or cured. The applicability and economic feasibility of using these methods in nursing homes needs to be determined.[37] Given current federal regulations on incontinence, many homes are planning to use similar programs with existing staff; done carefully and slowly, this shows great promise.

Summary

With the increasing evidence of efficacy of behavioral training and exercises, most older incontinent patients can avoid both surgery and medications for stress incontinence or detrusor instability. The primary care physicians' role should focus on screening all older patients for incontinence and providing a comprehensive evaluation to identify transient causes, medication effects, and functional problems. In the office, simple cystometry will reliably identify stress incontinence and exclude detrusor instability in women. Men need referral fairly frequently, because obstruction is difficult to exclude reliably. In institutional practice, nonurological causes are probably more common and consequently need to be aggressively diagnosed and treated. Most patients can be managed successfully without referral in this setting. Increasing availability of behavioral methods and exercise regimes will substantially improve incontinence in most patients.

Acknowledgments

The author thanks Nancy Hetmanski for expert manuscript preparation.

REFERENCES

1. Herzog AR, Fultz NH: Prevelance and incidence of urinary incontinence in community-dwelling populations. *J Amer Geriatr Soc* 1990; 38:273-81.
2. Williams ME, Pannill FC: Urinary incontinence in the elderly: Pathophysiology, diagnosis and treatment. *Ann Int Med* 1982; 97:895-907.
3. Wyman JF, Harkins SW, Choi SC, et al: Psychosocial impact of urinary incontinence in women. *Obstet Gynecol* 1987; 70:378-81.
4. Pannill FC: Practical management of urinary incontinence. *Med Clinic NA* 1989; 73:1423-39.
5. Ouslander JG: Geriatric urinary incontinence. *Disease A Month*, 38(2) February 1992.
6. Resnick NM, Yalla SV: Management of urinary incontinence in the elderly. *N Engl J Med* 1985; 313:800-5.
7. Urinary Incontinence Guideline Panel: Urinary incontinence in adults: Clinical practice guideline. Rockville: AHCPR Pub. No. 92-0038, 1992.
8. Diokno AC, Brock BM, Brown MB, Herzog AR: Prevelance of urinary incontinence and other urological symptoms in the noninstitutionalized elderly. *J Urol* 1986; 138:778-80.
9. Sier H, Ouslander J, Orzeck S: Urinary incontinence among geriatric patients in an acute care hospital. *JAMA* 1987; 257:1767-71.
10. Hu TW: Impact of urinary incontinence on health care costs. *J Amer Geriatr Soc* 1990; 38:292-5.
11. Resnick NM, Yalla SV, Laurino E: The pathophysiology of urinary incontinence among institutionalized persons. *N Engl J Med* 1989; 320:1-7.

12. Staskin DR, Zimmern PE, Hadley HR, Raz S: The pathophysiology of stress incontinence. *Urol Clinic NA* 1985; 12:271-278.
13. Pannill FC, Williams TF, Davis R: Evaluation and treatment of urinary incontinence in long-term care. *J Am Geriatr Soc* 1988; 36:902-10.
14. Boscia JA, Kabasa WB, Levison ME, et al: Lack of an association between bacteriuria and symptoms in the elderly. *Am J Med* 1986; 81:977-82.
15. Bergman A, Bhatia N: Urodynamics: Effect of urinary tract infection on urethral and bladder function. *Obstet Gynecol* 1985; 66:366-71.
16. Judge TG: The use of quinestradol in elderly incontinent women: A preliminary report. *Gerontol Clinica* 1969; 11:159-64.
17. Pannill FC: Diagnosis and treatment of urinary incontinence. In: Stults BM, Dere WH, ed. *Practical Care of the Ambulatory Patient.* Philadelphia: WB Saunders; 1989:281-92.
18. Ouslander JG, Leach GE, Staskin DR: Simplified tests of lower urinary tract function in the evaluation of geriatric urinary incontinence. *J Amer Geriatr Soc* 1989; 37:706-14.
19. Ouslander JG, Leach GE, Staskin DR, et al: Prospective evaluation of an assessment strategy for geriatric urinary incontinence. *J Amer Geriatr Soc* 1989; 37:715-24.
20. Ouslander JG, Leach GE, Abelson S, et al: Simple versus multichannel cystometry in the evaluation of bladder function in an incontinent geriatric population. *J Urol* 1988; 140:1482-86.
21. Diokno AC, Brown MB, Brock MB, et al: Clinical and cystometric characterisitics of continent and incontinent noninstitutionalized elderly. *J Urol* 1988; 140:567-71.
22. Pannill FC: Urinary Incontinence. In: Panzer RJ, Black ER, Griner PF, eds: *Diagnostic Strategies for Common Medical Problems.* Philadelphia: American College of Physicians. 1991; 420-9.
23. Raz S: Vaginal surgery for stress incontinence. *J Amer Geriatr Soc* 1990; 38:345-7.
24. Wells TJ, Brink CA, Diokno AC, et al: Pelvic muscle exercise for stress urinary incontinence in elderly women. *J Amer Geriatr Soc* 1991;39:785-91.
25. Burgio KL, Whitehead WE, Engel BT: Urinary incontinence in the elderly: Bladder-sphincter biofeedback and toileting skills training. *Ann Int Med* 1985; 104:507-15.
26. Stanton SL: Suprapubic approaches for stress incontinence. *J Amer Geriatr Soc* 1990; 38:348-51.
27. Burgio KL, Pearce KL, Lucco AJ: *Staying dry: A practical guide to bladder control.* Baltimore: Johns Hopkins University Press, 1989.
28. Wein AJ: Pharmacologic treatment of incontinence. *J Amer Geriatr Soc* 1990; 38:317-25 .
29. Hadley EC: Bladder training and related strategies for urinary incontinence in older people. *JAMA* 1986; 256:372-9.
30. Fantl JA, Wyman JF, McClish DK, et al: Efficacy of bladder training in older women with urinary incontinence. *JAMA* 1991; 265:609-13.
31. Ouslander JG, Blaustein J, Connor A, Pitt A: Habit training and oxybutynin for incontinence in nursing home patients: A placebo controlled trial. *J Amer Geriatr Soc* 1988; 36:40-46 .
32. McDowell BJ, Burgio KL, Dombrowski M, et al: An interdisciplinary approach to the assessment and behavioral treatment of urinary incontinence in geriatric outpatients. *J Amer Geriatr Soc* 1992; 40:370-4.
33. Gormley GJ, Stoner E, Bruskewitz RC, et al: The effect of finasteride in men with benign prostatic hypertrophy. *N Engl J Med* 1992; 327:1185-91.
34. Hu T, Igou JF, Kaltreider L, et al: A clinical trial of a behavioral therapy to reduce urinary incontinence in nursing homes. *JAMA* 1989; 261:2656-62.
35. Schnelle JF, Traughba B, Sowell VA, et al: Prompted voiding treatment of urinary incontinence in nursing home patients: A behavior management approach for nursing home staff. *J Am Geriatr Soc* 1989; 37:1051-7.
36. Colling J, Ouslander J, Hadley BJ, et al: The effects of patterned urge-response toileting (PURT) on urinary incontinence among nursing home residents *J Amer Geriatr Soc* 1992; 40:135-41.
37. Schnelle JF, Sowell VA, Hu T, et al: Reduction of urinary incontinence in nursing homes: Does it reduce or increase costs? *J Amer Geriatr Soc* 1988; 36:34-9.

UNIVERSITY OF MARYLAND
BALTIMORE

Delirium in Hospitalized Elderly Patients: Recognition, Evaluation, and Management

SHARON K. INOUYE, M.D.

> *"I've seen a dying eye*
> *Run round and round a room*
> *In search of something, as it seemed,*
> *Then cloudier become;*
> *And then, obscure with fog,*
> *And then be soldered down,*
> *Without disclosing what it be,*
> *'Twere blessed to have seen."*
> *Emily Dickinson*

Introduction

DELIRIUM, described as an acute disorder of attention and cognition, meets epidemiologic criteria of an important preventable disease.[1] First, it is a common problem, which may have serious complications, such as aspiration pneumonia, decubiti, functional decline, prolonged length of hospital stay, nursing home placement, and death. There is a subclinical form, and it is often unrecognized. Finally, delirium and its complications may be preventable.

Epidemiology.—Previous studies have estimated that the prevalence of delirium for elderly patients on admission to the hospital ranges from 10-40%. The incidence of new delirium developing during hospitalization has been estimated from 25-60%.[2-17] Hospital mortality estimates range from 10-65%, 2-20 times that of control patients without delirium,[18-26] which is as high as the mortality associated with myocardial infarction or sepsis. The reasons for the wide ranges in these estimates include variations in study populations examined, differing definitions of delirium, differing case-finding methods, varying age cutoffs, and risk groups, eg, medical, surgical, and intensive care populations.

Previous studies[27] have estimated that if the length of hospital stay of each acutely confused hospitalized elderly patient could be reduced by but one day, the savings to Medicare would amount to one to two billion dollars per year.

Definition.—The definitions and diagnostic criteria for delirium have continued to evolve. The Diagnostic and Statistical Manual of the American Psychiatric Association (DSMIII-R) provides standardized diagnostic criteria, which have been widely accepted.[28] A simplified, operationalized diagnostic algorithm was developed for the Confusion Assessment Method.[29] These diagnostic criteria are included in Table 1.

Recognition

Myths and realities.—Delirium is often unrecognized in older patients. Previous studies have found that from 32-66% of cases of delirium were unrecognized by the physicians caring for the patients.[27,30,31] There appear to be many reasons for this lack of recognition, which is the first and most crucial step in evaluation of delirium. First, there has been a genuine lack of awareness of delirium as an important medical syndrome, analogous to "chest pain" or "syncope." Moreover, clinicians have not been aware that delirium may be a medical emergency, and in the elderly, may be the sole manifestation of life-threatening illness. Delirium is often misdiagnosed as dementia, depression, or senescence (ie, part of the normal aging process). Finally, the fast pace and technologic focus of current hospital care allow scant opportunity for cognitive assessment of older patients. Very little time is spent at the bedside, providing little opportunity to observe

---

SHARON K. INOUYE, M.D., M.P.H., Assistant Professor of Medicine, Yale University School of Medicine.

Reprint requests to: Sharon K. Inouye, M.D., M.P.H., Yale-New Haven Hospital, 20 York Street, Tompkins 15, New Haven, CT 06504.

Grant support: In part by grants from the Retirement Research Foundation (Grants #90-44, 91-66), Sandoz Foundation for Gerontologic Research (Grant #11), and the American Federation for Aging Research. Dr. Inouye is a Dana Foundation Faculty Scholar and recipient of Academic Award #IK08AG00524-01 from the National Institute on Aging.

Table 1. Diagnostic Criteria for Delirium

DSM-III R Diagnostic Criteria[28]

- A. Reduced ability to maintain and shift attention to external stimuli
- B. Disorganized thinking, as indicated by rambling, irrelevant, or incoherent speech
- C. At least two of the following:
  1. Reduced level of consciousness
  2. Perceptual disturbances: misinterpretations, illusions, or hallucinations
  3. Disturbance of sleep-wake cycle with insomnia or daytime sleepiness
  4. Increased or decreased psychomotor activity
  5. Disorientation to time, place, or person
  6. Memory impairment
- D. Abrupt onset of symptoms (hours to days), with daily fluctuation
- E. Either one of the following:
  1. Evidence from history, physical examination, or laboratory tests of specific organic etiologic factor(s)
  2. Exclusion of nonorganic mental disorders when no etiologic organic factor can be identified

The Confusion Assessment Method (CAM) Diagnostic Algorithm[29]

Feature 1. **Acute Onset and Fluctuating Course**
This feature is usually obtained from a family member or nurse and is shown by positive responses to the following questions: Is there evidence of an acute change in mental status from the patient's baseline? Did the (abnormal) behavior fluctuate during the day, that is, tend to come and go, or increase and decrease in severity?

Feature 2. **Inattention**
This feature is shown by a positive response to the following question: Did the patient have difficulty focusing attention, for example, being easily distractible, or having difficulty keeping track of what was being said?

Feature 3. **Disorganized Thinking**
This feature is shown by a positive response to the following question: Was the patient's thinking disorganized or incoherent, such as rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject?

Feature 4. **Altered Level of Consciousness**
This feature is shown by any answer other than "alert" to the following question: Overall, how would you rate this patient's level of consciousness? (alert [normal], vigilant [hyperalert], lethargic [drowsy, easily aroused], stupor [difficult to arouse], or coma [unarousable]).

*The diagnosis of delirium by CAM requires the presence of features 1 and 2 and either (3 or 4).

cognitive changes during hospitalization. Table 2 outlines some of the myths and realities about delirium collected during interviews with housestaff and nursing staff over a two-year period at Yale-New Haven Hospital (Inouye SK: Unpublished observations). This table highlights some of the misconceptions about delirium and reasons for its under-recognition.

Simplified diagnostic criteria.—The lack of easily applied diagnostic criteria, which are understandable and usable by clinicians without psychiatric training, has also undoubtedly contributed to the recognition problem. Thus, the Confusion Assessment Method (CAM)[29] was created as a means to help nonpsychiatrically trained clinicians rapidly identify and recognize delirium (See Table 1). Most of the features of the Confusion Assessment Method can be assessed with a simple cognitive evaluation at the bedside. However, knowledge of the clinical course is essential to diagnose delirium, and this information must sometimes be obtained from a family member, caretaker, or an outside observer.

The CAM was tested in a two-site study involving 56 subjects, and was demonstrated to have a sensitivity from 94-100%, specificity from 90-95%, positive predictive accuracy of 91-94%, and negative predictive accuracy of 90-100%.[29]

Forms of delirium.—Clinically delirium can range from hyperactive delirium—where the patient appears agitated, hallucinating, and behaving inappropriately, to hypoactive delirium—where the patient appears lethargic and somnolent. In addition, patients can fluctuate along this spectrum of delirium. The elderly are particularly likely to present with the hypoactive or lethargic form of delirium, which is often unrecognized.

Delirium vs dementia.—A crucial difficulty in the diagnosis of delirium is distinguishing it from an underlying or long-standing confusional state, such as demen-

UNIVERSITY OF MARYLAND
BALTIMORE

Table 2.—Myth and Reality About Delirium in Hospitalized Elderly Patients

Myth	**Reality**
"Older people get confused in the hospital; it's to be expected."	Delirium is a potential medical emergency, and may be the sole manifestation of life-threatening illness.
"Oriented times one… this patient must have Alzheimer's disease."	Delirium is often misdiagnosed as dementia or depression, or misattributed to senescence. Cognitive function is rarely assessed, and prior cognitive status usually unknown.
"The hallmarks of delirium are agitation, disorientation, hallucinations, and inappropriate behavior."	More often, in older persons, delirium presents with lethargy and decreased activity—the hypoactive form of delirium, which is easily overlooked.
"The nurse says the patient got confused last night, but (s)he's totally with-it now. I don't think anything is going on."	Lucid intervals are characteristic of delirium. A fluctuating course is one of its key features.

tia. The differential diagnosis is crucial, because delirium is an acute medical emergency, and dementia carries a quite different prognosis. Delirium does occur superimposed on dementia in 1/3-1/2 of cases. Differentiating these two conditions is crucial but difficult. Some distinguishing features are listed in Table 3. These two conditions are differentiated by the acute onset, fluctuating course, inattention, and altered level of consciousness that are associated with delirium.

It is important to note that disorientation and memory impairment may be present with both conditions, and may be absent in delirium. Thus, they are not useful in differential diagnosis. However, because of the high prevalence of delirium in hospitalized elderly patients, any patient with a deterioration in mental status is best presumed to be delirious until proven otherwise.

Table 3.—Comparative Features of Delirium and Dementia

	Delirium *vs*	**Dementia**
Onset	Abrupt	Insidious
Duration	Hours to days	Months to years
Attention	Impaired	Normal unless severe
Consciousness	Fluctuating, reduced	Clear
Speech	Incoherent, disorganized	Ordered, anomic/ aphasic

Causes

Pathophysiology.—The exact pathophysiology of delirium remains unclear. Delirium has been called "acute brain failure," and a frequent finding has been generalized slowing by electroencephalography.[32-34] Thus, it is a global process, but the exact cellular and molecular mechanisms have not been determined. Most experts agree that it appears to be the common final pathway of many different pathogenic mechanisms. The hypothesized mechanisms have included: reduction in cerebral oxidative metabolism; decreased synthesis of neurotransmitters, such as acetylcholine; and hypercortisolemia, resulting in hypothalamic impairment.[35-40] Further studies are underway to clarify these underlying mechanisms.

Etiology.—Table 4 presents some of the important underlying etiologies of delirium, with the acronym DELIRIUM. Delirium appears to be the final common pathway of many different underlying etiologies. Many of these conditions are rather serious, and can be treatable. Thus, searching for the underlying cause(s) is essential.

Dementia is an important risk factor for delirium, with demented patients being at five to nine times increased risk for developing delirium in the hospital.[15,17] Electrolyte disorders, particularly hyper- or hyponatremia, and hypercalcemia are important etiologic factors. Insufficiency or failure of any major organ system can be an etiologic factor, including disorders of the lungs, liver, heart, kidney, and brain.[3,18,21,41-43] Both hypoxemia and hypercarbia may have been associated with the development of delirium, and clinicians must be aware of occult respiratory failure in the elderly, which may not present

Table 4.—Etiology

Dementia
Electrolytes
Lungs, liver, heart, kidney, brain
Infection
Rx
Injury, pain, stress
Unfamiliar environment
Metabolic

with the usual signs and symptoms of dyspnea or tachypnea.[44] Acute myocardial infarction or congestive heart failure can present primarily as altered mental status in an elderly patient, without the usual complaints of chest pain or shortness of breath.[44] Finally, any primary cerebral disorder, such as stroke, brain trauma, infection, or tumor, can present as delirium in an elderly patient.[18,21,41-43,45]

Occult infection is a particularly noteworthy cause of delirium.[18,21,41-43,45] Elderly patients may not mount the febrile or leukocytotic response expected with infection, and clinicians must assess carefully for an underlying pneumonia, urinary tract infection, abdominal abscess, or infected joint.[44] Drug or alcohol withdrawal is another important, often unsuspected, cause of delirium in the elderly.[21,42-43]

Injury, pain, psychologic stress, and unfamiliar environment can also contribute to delirium in hospitalized elderly patients. Environmental aspects, such as sleep deprivation, immobilization, and sensory overload or deprivation may be important aggravators in the hospital environment.[43,45-46] A variety of metabolic disorders, including thyroid disease, adrenal disorders, hyper- or hypoglycemia, may contribute to delirium.[21,41-43,45]

Medications are the most common reversible etiology of delirium in hospitalized elderly patients.[21,42,43,46,47] Medications associated with delirium are listed in Table 5. Particularly problematic medications for elderly hospitalized patients are long-acting benzodiazepines (Dalmane®, Valium®, Librium®), which should be avoided whenever possible. In addition, medications with anticholinergic side effects (eg, antihistamines, antidepressants, antipsychotics, and antispasmodics) commonly result in delirium in elderly patients. Many cases of delirium may be preventable through avoidance, substitution, or dose reduction of potentially psychoactive medications.

Evaluation

The cornerstone of evaluation of delirium includes a careful, detailed history and physical examination and cognitive assessment. The time-course of any mental status change should be established, and any potential clues to underlying etiology should be gleaned. Many brief, widely-used instruments are available for bedside cognitive assessment.[48-51] The clinician will need to be particularly astute to search for clues of an occult infectious process or focal neurologic changes.

Review of the medication list is a high-yield procedure, and should be carried out in every patient, including over-the-counter medications (a complete "medicine chest biopsy"). Further laboratory evaluation should be targeted and carefully tailored to the individual clinical situation. Laboratory tests (eg, complete blood count, electrolytes, blood urea nitrogen, creatinine, glucose, calcium, phosphorus, liver function tests) that search for occult infection (eg, urinalysis, chest roentgenogram, selected cultures), and that search for occult respiratory failure or cardiac disease (arterial blood gas, electrocardiogram) may be indicated as dictated by the individual clinical situation.

The use of brain imaging, lumbar puncture, or electroencephalography in the evaluation of delirium remains controversial. Clearly, cerebrospinal fluid examination must be carried out in the febrile delirious patient, whenever meningitis is suspected. However, in the afebrile patient with mental status change, where no new focal neurologic findings are present, the clinical yield of cerebrospinal fluid examination and brain imaging remains low.[52] In our clinical practice, lumbar puncture and brain imaging are carried out in less than 10% of patients who present with acutely altered mental status.

Management

Medical management.—In general, nonpharmacologic management of the acutely confused patient should be used whenever possible, such as: presence of family members, orienting influences, sitters, transferring the patient to a private room or closer to the nurse's station. Any medications to treat the agitation or hallucinations will invariably lead to further clouding of the mental status, and obscure efforts to follow the clinical course of the mental status change. If, however, the patient is a threat to his or her own safety or the safety of others, or if the delirium will result in the interruption of needed therapies (eg, intubation, intravenous line, nasogastric tube, bladder catheter, etc.), then treatment is recommended with haloperidol (Haldol®) or thioridazine (Mellaril®). The principles of drug therapy in the older patient are to "start low, go slow, and reassess frequently." Haloperidol has less orthostatic hypotension and anticholinergic side effects than thioridazine, and is available in parenteral form; however, it has a higher rate of dystonias and extrapyramidal side effects. Thioridazine has a higher rate of orthostatic hypotension and anticholinergic side effects, but is more sedating (which may be beneficial in extremely agitated patients). We recommend starting with haloperidol, 0.5 to 1.0 mgs orally or parenterally, or thioridazine, 10 to 20 mgs orally, then repeating the dose every 20-30 minutes as needed until sedation is achieved. The end-point should be an awake but manageable patient, not an obtunded patient. For the average elderly patient who has not previously been treated with an antipsychotic medication, a total of no more than 3 to 5 mgs of haloperidol, or 30 to 50 mgs thioridazine should be sufficient to achieve these end-points. The medication should then be repeated as needed, with careful titration and tapering of the dose over one to two days.

Hospital management.—Recommendations for ward management of delirium are outlined on Table 6.[53] Note that these same management strategies may be used in an elderly patient to prevent delirium from developing during hospitalization. Perhaps the most important intervention is to allow a period of uninterrupted sleep at night if at all possible. This may require coordination of medications, vital signs, intravenous fluids, and procedures to allow the patient to sleep. Particular care should be taken to make sure the lights are turned off, even when attending to a roommate, and to ensure decreased noise level at night, including hallway noise and conversations.

Attention to orienting influences in the environment is important. The room should have a clock and calendar to keep the patient oriented. Glasses, hearing aids, and dentures should be worn. Often, patients will come into the hospital without these, and family members should be instructed to bring them in as soon as possible. Obtain familiar possessions from home, such as bedside photos, religious items (eg, rosary beads, Bible), books, knitting equipment. Request that family members stay with the patient, as they can provide essential reorientation, effective communication, and support for the patient. If a regular routine or schedule of having family members present in shifts can be arranged, this is ideal. Consistent medical and nursing staff should be in attendance, to provide continuity and reorientation for the patient.

Communication is essential for the care of the confused elderly patient, but is sometimes difficult because of hearing impairment, cognitive impairment, or language difficulties. Face-to-face contact is essential; encourage a give-and-take exchange with the patient. Allow the patients to participate in decision-making about their own care.

The patient should be encouraged to maintain his or her usual activity level in the hospital. All efforts should be made to avoid physical restraints and/or bed-rest orders, both of which have been shown to increase the risk of delirium and functional decline in hospitalized elderly patients. The patient should be encouraged to participate in self-care and other personal activities to maximize mobility and independence.

Table 5.—Drugs Associated with Delirium in the Elderly

- Sedatives/hypnotics
  - Benzodiazepines [especially Dalmane® (flurazepam), Valium® (diazepam), Librium® (chlordiazepoxide)]
  - Barbiturates
  - Sleeping medications (chloral hydrate)
- Narcotics
- Anticholinergics
  - Antihistamines (diphenhydramine, hydroxyzine)
  - Antispasmodics (belladonna, diphenoxylate)
  - Heterocyclic antidepressants (amitriptyline, doxepin)
  - Neuroleptics (chlorpromazine, haloperidol, thioridazine)
  - Antiparkinsonian (benztropine, trihexyphenidy)
- Cardiac
  - Digitalis glycosides
  - Antiarrhythmias
  - Calcium-channel blockers
  - Antihypertensives (beta-blockers, methyldopa, reserpine)
- Gastrointestinal
  - H_2 antagonists (cimetidine, ranitidine)
  - Metoclopramide (Reglan®)
- Miscellaneous
  - Corticosteroids
  - Nonsteroidal antiinflammatory drugs and salicylates
  - Anticonvulsants
  - Levodopa
  - Lithium
- Over-the-counter drugs
  - Cold/sinus preparations (antihistamines, pseudoephedrine)
  - Sleep aids (diphenhydramine, alcohol-containing elixirs)
  - Stay-awake (caffeine)
  - Nausea/motion sickness (Donnagel®, meclizine)
  - Weight loss (phenylpropanolamine)

Conclusions

In conclusion, delirium is a common and serious problem for hospitalized elderly patients. Recognition may be difficult, and requires a heightened awareness of the importance of cognitive evaluation in older patients. Knowledge of the clinical course of mental status change is essential to diagnose delirium, and this information

Table 6.—Ward Management of Delirium*

Environment Sensory input: not excessive, inadequate, or ambiguous. Room should have adequate light and be quiet. Some patients prefer radio or television for familiar background stimulation. Present one stimulus or task at a time. Medication schedules should not interrupt sleep.
Orientation Room should have a clock, calendar, and chart of the day's schedule. Keep the patient in the same surroundings. Verbal reminders of the time, day, and place should be used frequently. Evaluate the need for eyeglasses, hearing aids, and foreign language interpreters.
Familiarity Obtain familiar possessions from home to help orient the patient, particularly objects at the home bedside. Request family members to stay with the patient. They provide the basis for orientation, effective communication, support, and aftercare planning. Discuss familiar areas of interest, eg, hobbies, occupation. Allow the same staff members to consistently care for the patient.
Communication Instructions and explanations should be clear, slow-paced, simple, and repetitive. Use face-to-face contact. Convey an attitude of warmth and kind firmness. Consistently address the patient as he or she prefers. Begin each contact with orienting and identifying information. Acknowledge the patient's emotions and encourage verbal expression.
Activities Avoid physical restraint. Allow free movement, provided the patient is safe. Encourage self-care and other personal activities to reinforce competence and enhance self-esteem.

*Adapted from Beresin E.[53] Reproduced with permission.

must often be obtained from an outside observer, such as a family member or caretaker. Delirium is often overlooked or misdiagnosed as dementia, depression, or benign senescence. The hypoactive, lethargic form of delirium is particularly common in older patients, and is easily overlooked.

Although the exact pathophysiology of delirium remains unclear, delirium appears to be the final common pathway of a variety of underlying etiologies. Delirium has been called "the great nonspecific reaction of older people."[54] Many of the underlying causes are serious, but are treatable; thus, it is important to look carefully for the underlying cause. In addition, delirium is often of multifactorial etiology, and several factors may be contributing simultaneously or in a cumulative fashion.

Many cases of delirium may be preventable, especially through avoidance of psychoactive medications wherever possible, and minimization of environmental disruption for the elderly patient. Pharmacologic management of the delirious patient, eg, with major tranquilizers, is recommended only when the patient is a danger to him or herself or to others.

Acknowledgments

The author thanks Geraldine Hawthorne for manuscript preparation.

REFERENCES

1. Mausner JS, Kramer S: *Epidemiology: An Introductory Text.* 2nd ed. Philadelphia: WB Saunders, 1974: 217-30.
2. Rosin AJ, Boyd RV: Complications of illness in geriatric patients in hospital. *J Chron Dis* 1966; 19:307-13.
3. Hodkinson HM: Mental impairment in the elderly. *J R Coll Physicians Lond* 1973; 7:305-17.
4. Bergman K, Eastham EJ: Psychogeriatric ascertainment and assessment for treatment in an acute medical ward setting. *Age Ageing* 1974; 3:174-88.
5. Seymour DG, Henschke PJ, Cape RDT, et al: Acute confusional states and dementia in the elderly: The role of dehydration/volume depletion, physical illness and age. *Age Ageing* 1980; 9:137-46.
6. Chisholm SE, Deniston OL, Ingrisan RM, et al: Prevalence of confusion in elderly hospitalized patients. *J Gerontol Nurs* 1982; 8:87-96.
7. Gillick MR, Serrell NA, Gillick LS: Adverse consequences of hospitalization in the elderly. *Soc Sci Med* 1982; 16:1033-8.

8. Warshaw GA, Moore JT, Friedman SW, et al: Functional disability in the hospitalized elderly. *JAMA* 1982; 248:847-50.
9. Cavanaugh S: The prevalence of emotional and cognitive dysfunction in a general medical population: Using the MMSE, GHQ, and BDI. *Gen Hosp Psychiatry* 1983; 5:15-24.
10. Williams MA, Campbell EB, Raynor WJ, et al: Predictors of acute confusional states in hospitalized elderly patients. *Res Nurse Health* 1985; 8:31-40.
11. Fields SD, MacKenzie CR, Charlson ME, et al: Reversibility of cognitive impairment in medical inpatients. *Arch Intern Med* 1986; 146:1593-6.
12. Cameron DJ, Thomas RI, Mulvihill M, et al: Delirium: A test of the Diagnostic and Statistical Manual III Criteria on medical inpatients. *J Am Geriatr Soc* 1987; 35:1007-10.
13. Foreman MD: Confusion in the hospitalized elderly: Incidence, onset, and associated factors. *Res Nurs Health* 1989; 12:21-9.
14. Rockwood K: Acute confusion in elderly medical patients. *J Am Geriatr Soc* 1989; 37:150-4.
15. Francis J, Martin D, Kapoor WN: A prospective study of delirium in hospitalized elderly. *JAMA* 1990; 263:1097-101.
16. Johnson JC, Gottlieb GL, Sullivan E, et al: Using DSM-III criteria to diagnose delirium in elderly general medical patients. *J Gerontol* Medical Sciences. 1990; 45:113-9.
17. Schor JD, Levkoff SE, Lipsitz LA, et al: Risk factors for delirium in hospitalized elderly. *JAMA* 1992; 267:827-31.
18. Roth M: The natural history of mental disorder in old age. *J Ment Sci* 1955; 101:281-303.
19. Kay DWK, Norris V, Post F: Prognosis in psychiatric disorders of the elderly: An attempt to define indicators of early death and early recovery. *J Ment Sci* 1956; 120: 129-40.
20. Bedford PD: General medical aspects of confusional states in elderly people. *BMJ* 1959; 2:185-8.
21. Guze SB, Cantwell DP: The prognosis in "organic brain" syndromes. *Am J Psychiatry* 1964; 120:878-81.
22. Guze SB, Daengsurisri S: Organic brain syndromes: Prognostic significance in general medical patients. *Arch Gen Psychiatry* 1967; 17:365-6.
23. Rabins PV, Folstein MF: Delirium and dementia: Diagnostic criteria and fatality rates. *Br J Psychiatry* 1982; 140:149-53.
24. Weddington WW: The mortality of delirium: An underappreciated problem? *Psychosomatics* 1982;23: 1232-5.
25. Black DW, Warrack G, Winokur G: The Iowa record-linkage study II. Excess mortality among patients with organic mental disorders. *Arch Gen Psychiatry* 1985; 42:78-81.
26. Fields SD, MacKenzie CR, Charlson ME, et al: Cognitive impairment: Can it predict the course of hospitalized patients? *J Am Geriatr Soc* 1986; 34:579-85.
27. Levkoff SE, Besdine RW, Wetle T: Acute confusional states (Delirium) in the hospitalized elderly. *Ann Rev Gerontol Geriatr* 1986; 6:1-26.
28. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed, rev. Washington, DC: Am Psychiatric Assoc; 1987:97-124.
29. Inouye SK, van Dyck CH, Alessi CA, et al: Clarifying confusion: The Confusion Assessment Method; A new method for detection of delirium. *Ann Intern Med* 1990; 113:941-8.
30. Francis J, Strong S, Martin D, Kapoor W: Delirium in elderly general medical patients: Common but often unrecognized. *Clin Res* 1988; 36:711A.
31. Williams MA, Holloway JR, Winn MC, et al: Nursing activities and acute confusional states in elderly hip-fractured patients. *Nurs Res* 1979; 28:25-35.
32. Romano J, Engel GL: Delirium I: Electroencephalographic data. *Arch Neurol Psychiatry* 1944; 51:356-77.
33. Pro JD, Wells CE: The use of the electroencephalogram in the diagnosis of delirium. *Dis Nerv Sys* 1977; 38:804-8.
34. Trzepacz TT, Brenner RP, Coffman G, et al: Delirium in liver transplantation candidates: Discriminant analysis of multiple tests variables. *Biol Psychiatry* 1988; 24:3-14.
35. Lipowski AJ: Delirium: *Acute Confusional States.* Oxford: Oxford University Press; 1990:141-74.
36. Engel GL, Romano J: Delirium, a syndrome of cerebral insufficiency. *J Chronic Dis* 1959; 9:260-77.
37. Blass JP, Gibson GE, Duffy TE, et al: Cholinergic dysfunction: A common denominator in metabolic encephalopathies. In: Pepeu G, Ladinsky H, eds. *Cholinergic Mechanisms* New York: Plenum Press; 1981:921-8.
38. Siesjo BK, Carlsson C, Hagerdal M, et al: Brain metabolism in the critically ill. *Crit Care Med* 1976; 4:283-94.
39. McIntosh TK, Bush HL, Yeston NS, et al: Beta-endorphin, cortisol and postoperative delirium: A preliminary report. *Psychoneuroendocrinology* 1985; 10:303-13.
40. Kral VA: Stress and mental disorders of the senium. *Med Serv J* (Canada) 1962; 18:363-70.
41. Flint FJ, Richards SM: Organic basis of confusional states in the elderly. *Brit Med J* 1956; 2:1537-9.
42. Sirois F: Delirium: 100 cases. *Can J Psychiatry* 1988; 33:375-8.
43. Purdie FR, Honigman B, Rosen P: Acute organic brain syndrome: A review of 100 cases. *Ann Emerg Med* 1981; 10:455-61.
44. Rosenthal G, Steel K: Difference in the presentation of disease. In: Hazzard WR, Andres R, Bierman EL, Blass JP, eds. *Principles of Geriatric Medicine and Gerontology* 2nd ed. New York: McGraw-Hill, 1990: 212-7.
45. Koponen H, Partanen J, Paakkonen A, et al: EEG spectral analysis in delirium. *J Neurol Neurosurg Psychiatry* 1989; 52:980-5.
46. Millar HR: Psychiatric morbidity in elderly surgical patients. *Br J Psychiatry* 1981; 138:17-20.
47. Moses H, Van Kaden BA: Neurologic consultation in a general hospital: Spectrum of iatrogenic disease. *Am J Med* 1986; 81: 955-8.
48. Folstein MF, Folstein SE, McHugh PR: The Mini-Mental State Examination: A practical method for grading the cognitive state of patients for the clinician. *J Psychiatry Res* 1975; 12:189-98.
49. Pfeiffer E: A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. *J Am Geriatr Soc* 1975; 23:433-41.
50. Jacobs JW, Bernhard MR, Delgado A, et al: Screening for organic mental syndromes in the medically ill. *Ann Intern Med* 1977; 86:40-6.
51. Nelson A, Fogel BS, Faust D: Bedside cognitive screening instruments: A critical assessment. *J Nerv Ment Dis* 1986; 174: 73-83.
52. Wolfson L, Katzman R: The neurologic consultation at age 80. In: Katzman R, Rowe JW, eds. *Principles of Geriatric Neurology* Philadelphia: FA Davis; 1992:84-5.
53. Beresin EV: Delirium in the elderly. *J Geriatr Psychiatry Neurol* 1988; 1:127-43.
54. Arie T: Confusion in old age. *Age Ageing* 1978; 7(supp):72-6.

Fairfield County Medical Association
Hartford County Medical Association
New Haven County Medical Association
New London County Medical Association
CNA Insurance Companies

We're proud of the company we keep

© 1993 Poe & Associates, Inc.

Since 1981, each of these important associations has endorsed and supported the Physicians Protector Plan®, the finest professional liability insurance program available in Connecticut today.

This 12-year working relationship among the medical community, the program administrator, and CNA Insurance Companies has provided physicians in Connecticut with unsurpassed commitment, expertise, financial stability, and a stable professional liability environment.

We at PHYSICIANplans® are proud to be associated with Connecticut's medical community and with CNA. If you're not currently participating in this fine program this is an excellent opportunity to discuss how it can benefit you and your practice. Give us a call.

1-800-533-4539

PHYSICIANplans®

CNA
For All the Commitments You Make®

The Physicians Protector Plan® is a trademark of Poe & Associates, Inc., Tampa, Florida, and is underwritten by the Continental Casualty Company and National Fire Insurance Company of Hartford, two of the CNA Insurance Companies.

UNIVERSITY OF MARYLAND
BALTIMORE

Physical Examination of the Aged Patient

HENRY SCHNEIDERMAN, M.D.

ABSTRACT—The aged patient presents challenges to the examining physician. Some of these relate to inefficient historical information transfer, reduced mobility, increased disease burden, cognitive and sensory alterations, and confusing background changes in the prevalence and interpretation of physical signs that would be unequivocally abnormal in the younger adult. This review suggests techniques to overcome these difficulties. Descriptions of methods of improving evaluation and interpretation are grouped into the following categories: general appearance and vital signs; skin; head and neck; respiratory and cardiovascular systems; abdomen, rectum and genitalia; musculoskeletal system; hands and feet; and neurologic and mental status evaluations. Several newly recognized signs are also described.

The extra time, effort and ingenuity required for examination of the aged patient are richly repaid by enhanced diagnosis.

Introduction

Bedside assessment of the multiproblem aged patient, particularly the physically frail and mentally impaired, offers a unique challenge disguised as a daunting hurdle. To meet this challenge, the clinician must prioritize, persist, employ patience, and keep numerous variables in mind, while distinguishing noise from signal

The database outlined below is achievable only with practice and with expenditure of time and effort on each case. New analyses of bedside diagnosis[1] can be applied to geriatric evaluation to raise its high yield, while discarding techniques that consume time without illuminating. Although the principal focus here is physical examination, the history is so crucial that some attention must first be devoted to it.

Taking the History

An elderly patient often tires during a protracted session; the initial evaluation may need to be divided into two sessions or even more.

Many elderly patients have sensory deficits (such as hearing loss), aphasia, or mental incapacity. Other information sources include family members, a home health aide, or medical records. Unfortunately, the perceptions and concerns of a caregiver often differ from those of the patient. The family's concerns may focus on symptoms that disrupt the household or upset the observer. There is a tendency for the spouse to underestimate cognitive loss. A spouse may preempt errors and revise mistaken statements not only in the history, where the corrected data are essential, but also on cognitive testing. In such instances, one must gently instruct against answering for the patient. Advance review of medical records represents time well spent. A comprehensive Problem List contributes to effective focus both at this point and afterward.

To reduce difficulties relating to hearing loss, the site of examination should be free of extraneous noise. The physician moves close to the patient, faces him directly and speaks slowly. A deep (low-pitched) voice may help overcome high-frequency hearing loss. If the patient still can't hear, examine the auditory canals for cerumen. The patient should wear dentures, eye-glasses, and hearing aid to facilitate both verbal and nonverbal communication. The room must be well lighted.

It is essential to determine reliability early. This may require brief mental status evaluation near the outset, introduced so that the patient will not be offended: "I will ask some questions to test your memory and concentration, as with every new patient in my practice."

HENRY SCHNEIDERMAN, M.D., Associate Professor of Medicine (Geriatrics) and Pathology, University of Connecticut School of Medicine.

Address for correspondence, proofs and reprint requests: Henry Scheiderman, M.D., University of Connecticut School of Medicine, 263 Farmington Avenue, Farmington CT 06030-3940, (203) 679-4211.

Many aged people have a lengthy medical history, and understandably experience difficulty about details. One's educated guesses about prior surgery, based on location of surgical scars on physical examination (especially for thyroidectomy, sternotomy, cholecystectomy, and total hip replacement) may jog the patient's memory—and build the relationship as one displays interest, astute observation, and sound inference.

In taking a medication history, one must specify vitamins, mineral supplements, laxatives, minor pain medicines, nonprescription sleep aids, and cold-and-flu remedies. Otherwise, patients do not bother to mention them, yet their medical consequences can be profound. For example, over-the-counter cold remedies can precipitate both urinary retention and cognitive dysfunction in susceptible persons. Topical drugs are reviewed as well. The patient can be asked to bring along a shopping bag containing every medicine and health product in the house—not only those in the medicine cabinet.

A screening question for emotional trouble is, "Do you often feel sad or depressed?"[2] In seeking an exogenous cause, ask about recent loss of a consort, friend, even a pet; of income, as through retirement; or of home, status, or independence. Depression should always suggest the risk of suicide.

Physicians have often failed to elicit important functional and diagnostic information in the aged, including sensory impairment, both sight and hearing; memory loss; and urinary or fecal incontinence.[3] A simple wording for incontinence screening is, "Do you ever lose your urine and get wet?"

The family's ability to assist the patient is essential to know. A screening question is, "Who would be able to help you in case of illness or emergency?" The social history also covers assessment of living arrangements: number of rooms, elevators, adequacy of heating. A home visit is optimal, but without this one can ask, "Do you have trouble with stairs in your home? With getting into or out of the tub? With slipping on rugs? With poorly lit areas?" Nutrition, another area of frequently missed diagnoses[4] must be considered in relation to diverse social, economic, and functional factors that can impair intake.[5] Alcoholism is also underrecognized in the aged; questions about alcohol, tobacco, and other drugs round out this section.

The Activities of Daily Living (ADLs) and Instrumental Activities of Daily Living (IADLs) address mobility and self-sufficiency.[6] Questions may include, "Can you get out of bed yourself? Dress yourself? Make your own meals? Do your own shopping?" Ambulation may require the assistance of a cane, walker, or another person. The ability to bathe and toilet oneself, and independence in or the help of others for cleaning and housekeeping, maintaining one's own finances, and either driving or getting about on public transportation constitute additional useful information.

The physician, rather than a surrogate, needs to discuss and document the patient's wishes regarding cardiopulmonary resuscitation and mechanical ventilation for himself or herself. This should be undertaken only after some rapport exists late in the interview, or at a second visit. Related issues include intensive care unit utilization in the present state of health, and if cognitive impairment or a fatal illness were to develop. Any proxy designated in case of incapacity should be named and recorded at this session. The importance of a Living Will should be stressed. Concern that this *Values History* will frighten the patient is seldom warranted. The more matter-of-fact the physician is in approaching these questions, the easier it will be for the patient. Forms are available to simplify both discussion and recording.

Physical Examination Techniques

The examination is best conducted in a pleasant environment kept at 24° C to avoid chilling a disrobed old person. If an examining table is used, it is lowered to a height that facilitates transfers. A wide footstool with a stable armrest is close by to ease transfers. The patient is never left alone on the table, since a fall from the table can wreak havoc. A pillow should be available. Examining gowns are knee-length so they do not trip the patient. The patient will require additional time to undress, and to transfer to the examining table. Rushing is counterproductive: the inevitable response, further anxiety, creates further delay.

If the examination is conducted in the patient's room—for example, in a nursing home—and a roommate is present, bed-curtains are drawn or the roommate is asked to leave. The patient may choose whether or not to have a relative or aide in the room during the examination.

General appearance and vital signs.—It is striking how little is known about level of consciousness as a metabolic marker. Even the absence of narcosis precluding marked carbon dioxide retention has been challenged with a well-documented, glaring exception.[7]

Ongoing assessment of function begins right away, as clothing and hygiene are observed, along with the presence of a urinary catheter or adult diaper. If the patient is examined at the bedside, a water mattress, a sheepskin, or restraints should be noted.

Serial measurements of height and weight are most useful. Stable weight may reflect not only caloric balance but also the opposing effects of malnutrition and fluid retention.[5] Loss of height is a late sign in osteopenia.

With a slow peripheral pulse or any irregularity, a recount by auscultation at the cardiac apex is essential.

Recording the specific temperature is essential: the descriptor "afebrile" needs to be avoided, because it does not discriminate between hypothermia and normal temperature. Baseline measurement improves the recognition of fever: important temperature elevations may include any rise of 1.33° C (2.4° F) over baseline.[8] Thus if the basal temperature is not the "normal" 37.0° C, but 36.3° C, then a temperature of 37.6° C (99.7° F) that would otherwise be dismissed as trivial, may represent significant fever! Oral temperature ought not be measured immediately after consumption of cold liquids. Rectal temperatures are more accurate but more unpleasant for the patient. The diagnosis of hypothermia will be missed if one's instrument does not measure low temperatures, or if one assumes that failure to register a temperature means that the equipment is dysfunctional!

Since blood pressure (BP) fluctuates considerably, multiple measurements under resting conditions are requisite to a diagnosis of hypertension. BP may be overestimated in elderly patients because of arterial stiffness. This *pseudohypertension* is suspected if the brachial or radial artery remains palpable after the BP cuff has been inflated to a point greater than the systolic pressure (Osler's sign), that is, if the wall of the now nonpulsatile artery can still be felt after cuff inflation has ablated Korotkoff sounds.

Atrial fibrillation renders measurements of BP less accurate, and despite the high prevalence of this arrhythmia in the elderly, a standardized method of measuring the BP atrial fibrillation has not been established, let alone accepted .[9]

Routine assessment of pulse and blood pressure in two positions permits recognition of orthostatic hypotension, which is found in some normal as well as some symptomatic elders. Orthostatic hypotension consists of a drop in systolic BP of at least 15 mm Hg, or a fall in diastolic BP of at least 10 mm Hg, on being seated after a supine measurement, or on standing after a seated measurement. Its equivalent, orthostatic tachycardia, is a rise in pulse rate of greater than 16 beats per minute (a rise of four or more beats on the usual 15-second radial palpation) upon standing upright. To test for orthostatic findings, both pulse and BP are measured in the lower position, then again immediately after assuming the more upright posture, and still again after three minutes in this position. The test is terminated if the patient is uncomfortable, has chest pain, or becomes presyncopal. If the change from supine to seated discloses orthostatic hypotension, the upright measurement is not performed.

Postural hypotension with an unchanging pulse rate indicates autonomic dysfunction. A postprandial decrease in BP with virtually no compensatory increase in pulse rate has been demonstrated in elderly institutionalized patients.

The normal respiratory rate in the aged is 12 to 20 breaths per minute. A rate above some 22 breaths/min suggests the possibility of a lower respiratory tract infection, even prior to the appearance of other clinical signs, and before the emergence of a previously undifferentiated problem as respiratory.

Skin.—With aging, the skin wrinkles and becomes drier and more rough-surfaced. Cigarette smoking and excessive solar exposure amplify these effects. Since the dermis becomes thinner with age, ecchymoses may occur easily in traumatized areas of the skin, such as the forearm. Unexplained bruises may reflect this innocuous phenomenon, perhaps exacerbated by aspirin or anticoagulant therapy, or may offer a more ominous clue to elder abuse.

Loss of skin turgor is frequent in aging, so that this sign cannot be relied upon to corroborate an impression of intravascular volume depletion. It has been suggested that the sun-protected skin of the buttocks remains a valid indicator for this purpose, but research is lacking. (Nor do sunken eyes reliably signify dehydration.)

For the patient at risk, skin examination should include a search for pressure ulcers. The size of a pressure ulcer on the skin surface underestimates the extent of the subjacent lesion. Full turning of the bedbound patient onto the side is necessary, so that all lumbosacral, coccygeal, and perigluteal recesses can be brought into view by unfolding them. There is always erythema at the points on which one has been lying. However, erythema that cannot be blanched by pressure from the examining finger denotes the earliest recognizable bedsore (Shea grade I pressure ulcer). Skin at risk includes all promontories, so that the scapular tips, elbows, backs of ears, occiput, heels, malleoli, and greater trochanters share this propensity with the familiar low-back-and-buttocks combination.

Melanoma is suspected, rather than the usual nevi and seborrheic keratoses, when a lesion is larger than 6 mm across; variegated in color and pigmentation; shows deep black areas; has irregular borders and shape; or otherwise appears strange. Of special import in the aged is Hutchinson's freckle, melanoma *in situ* on the face. These large patches can extend laterally and ultimately can become deeply invasive and fatal. Any dark macule-patch wider than a centimeter on the face deserves dermatologic referral for evaluation and possible biopsy.

Head and neck.—The superficial temporal arteries, just anterior to the ears, are normally pulsatile, nontender, and not nodular, even in the very aged.[10] Giant-cell arteritis can change any of these characteristics except locale. In addition, scalp tenderness and nodules can be signs of giant cell arteritis. None of these features are sensitive indicators, but several are highly specific.[11]

HEALTH SCIENCES LIBRARY

Thus tenderness or absent pulsation in a temporal artery, or scalp tenderness or nodules, strongly suggests the disease, using the "SpPin" mnemonic of Sackett;[1] by contrast, the lack of sensitivity means that absence of these features does not rule out the condition .[1]

Xanthelasma palpebrarum and corneal arcus lose diagnostic importance with age, and need not be sought.

Vision testing with a Snellen chart or Jaeger card, while the patient wears his eyeglasses, is a valuable screening tool; check briefly to see that the glasses are properly centered and the lenses clean!

Fundus examination may be impossible without the pharmacologic help of one drop of tropicamide, instilled sterilely in each eye one half-hour before funduscopy. Prior to dilating the pupils this way, the depth of the anterior chamber is estimated by shining a light from the temporal side of the eye across the plane of the iris. If the entire plane is illuminated, anterior chamber depth is adequate, and the risk of precipitating acute angle-closure glaucoma is negligible. If, however, the nasal aspect of the iris is shadowed, the anterior chamber is narrowed, and then pupillodilation should be done only by an ophthalmologist if at all.

To screen hearing, a short question is whispered into each ear, while the examiner's lips are not in view: "What is your first name?" and "What's your last name?" are two simple ones even for the cognitively impaired.[12] Only with hearing loss or other otic symptoms is it necessary to perform otoscopy: removable cerumen is sought in the external auditory canal. If the patient performs poorly despite a hearing aid, it should be removed and inspected for wax that has plugged the ear mold and plastic tubing. If the battery is dead, a whistle (feedback) will not be heard when the volume is turned up. The familiar Weber and Rinné tests, wastes of time in persons with normal hearing, help localize hearing loss and discriminate sensorineural from conductive loss.

The mouth is examined for bleeding, purulent gums, broken teeth or carious roots, and candidosis. In seeking oral cancers in the high-risk elder—one who smokes tobacco and uses alcohol—the earliest recognizable form is a red focus, *erythroplasia*, that persists after two weeks of conservative hygienic and anti-infective measures, usually undertaken in collaboration with a dentist.[13]

The tongue may increase in size in an edentulous patient, producing a sort of *macroglossia ex vacuo*. Tongue enlargement also takes place in amyloidosis, acromegaly, and hypothyroidism. The dorsum is checked for papillary atrophy (beefy tongue; smooth, painful tongue), found in some cases of B vitamin deficiency including both riboflavin and B12. With tongue claudication, which is more specific for temporal arteritis than the better-known jaw claudication, but far less sensitive,[11] one looks for ischemic blanching at rest or with exercise, and for frank ischemic necrosis![14] The tongue is unsuitable for assessing volume status, because it is artificially dried in mouth-breathers, and because many aged persons have salivary insufficiency, with or without frank sicca (Sjögren's) syndrome, that produces chronic false-positive results. Air drying effect can be circumvented by inspecting the buccolabial fold instead of the dorsum of the tongue, but xerostomia cannot.

Numerous irritative conditions may occur around or under dentures. Examination of the patient with dentures must include looking at the oral mucosae after denture removal.

Meningeal signs show poorer positive *and* negative predictive values in the aged than in younger adults. Acute bacterial meningitis may lack these signs. Some of the same meningeal signs are present in a few aged persons without meningitis. Unfortunately the demented patient, in whom one relies most on signs, is the most likely to have these false-positive findings.[15]

Thyroid examination in the elderly can be simplified because of loss of fat and sternomastoid mass. Humility is essential, however, since most aged persons with thyroid disorders have normal neck examinations.

Respiratory and cardiovascular systems.—Tactile vocal fremitus can provide more information in a thinner, less muscled chest wall that transmits airway vibrations better. The confounding effect of chest wall thickness is frequently reduced in the elderly, so much so that percussion becomes the bedside method of choice to detect pulmonary consolidation in the demented individual who will not take deep breaths through the mouth no matter how creatively asked to do so. The more routinely that percussion is practiced, the more reliable it becomes. In some demented persons who cannot cooperate at all, the radiograph must assume primacy.

Pulmonary crackles occur in some normal aged persons. Correlations with symptoms and with other signs over time, as well as with chest radiographs, help distinguish innocent from pathologic crackles.

Late-onset asthma can be the cause of wheezing heard in an aged person. Another differential diagnosis is cardiac failure manifest as cardiac asthma. Serial quantification of forced expiratory time in aged persons with obstructive airway disease helps assess severity and response to therapy. By contrast, seeking forced expiratory wheezes should be abandoned because of lack of diagnostic utility.

Premature heartbeats and skipped beats are common, as is atrial fibrillation, recognized by an absolutely irregular *apical* heart beat, usually with a variable intensity of the first heart sound (S1).

The second heart sound (S2) provides a screening test for heart disease. It is ausculted at the base throughout several respiratory cycles, and briefly at the apex. S2 is single in most normal elders. If it is split, one seeks features of normality: splitting only in inspiration, splitting audible only along the left sternal border, and with the earlier aortic component being louder than the later pulmonic component. Reversed splitting, splitting that is present in expiration but wider in inspiration, a pulmonic component audible at the apex or louder than the aortic component, all speak to cardiorespiratory disease. Right bundle branch block can cause continuous, nonfixed splitting. Left bundle branch block among other conditions produces paradoxical (reversed, purely expiratory) splitting. Since activation of the right ventricle occurs before that of the left with a functioning artificial pacemaker, paradoxical splitting is expected in this setting if the pulmonic component can be heard at all.

Fourth heart sounds are common in normal aged persons. By contrast, third heart sounds are specific for heart disease. However, they are not sensitive markers for cardiac failure.

A common systolic murmur in the elderly is due to hemodynamically innocuous "aortic sclerosis." However, classical signs are often lacking in persons over 60 with aortic stenosis . The murmur is often unimpressively soft, the *pulsus parvus et tardus* lacking, and the pulse pressure not narrowed. Hence aortic stenosis may lurk in the guise of aortic sclerosis.

Besides these two conditions, mitral regurgitation, tricuspid regurgitation, pulmonic flow murmurs, and hypertrophic cardiomyopathy can all produce systolic murmurs. The locale of maximal intensity, pattern of radiation, and character of sound are principal means of distinguishing these. In addition, the murmur of hypertrophic cardiomyopathy is often intensified by Valsalva's maneuver, as is mitral regurgitation due to mitral valvular prolapse; all the others mentioned are not.

Asymptomatic carotid arterial bruits are markers of widespread rather than local atherosclerosis: they are associated as much with increase in stroke contralateral as ipsilateral to the bruit! Carotid bruits in the aged frequently disappear with no clinical event.[16] Totally obstructed carotid arteries are silent, so the absence of a bruit is not reassuring.

Palpation for silent, asymptomatic abdominal aortic aneurysms has diagnostic sensitivity in thin men, but not in obese men.[17] The absence of palpable foot pulses in an asymptomatic aged person is no cause for alarm if the foot is reasonably warm, and free of trophic changes. With ischemic ulceration, however, absence of foot pulses denotes a decreased chance of healing without revasculariztion .[18]

Difficulties with the interpretation of blood columns in the jugular veins of the aged include invisibility in thick necks; false-positive jugular venous distention when an uncoiled aorta has impaired venous outflow from the neck; and misleadingly flat neck veins obliterated by fibrosis. Light pressure just above the clavicle should cause a blood column to appear in the ipsilateral external jugular within seconds in every person with a patent vein, regardless of intravascular volume status. If this does not happen, the other side must be tested. If a blood column seems unduly tall, one can compare it with the other side to see which changes appropriately with position and breathing. The physiologic response is for the meniscus to fall with inspiration. The reverse constitutes Kussmaul's sign, which is seen in right ventricular myocardial infarction as well as in pericardial constriction.

Much leg edema due to venous insufficiency is misattributed to cardiac failure. To diagnose venous valve incompetence in the legs, the ambulatory patient is made to stand, so gravitational filling will be maximal.

Abdomen, rectum, and genitalia.—In old age the abdominal musculature becomes weaker and therefore less protective. Hence assessment of intraperitoneal pathology by palpation grows easier. The differential diagnosis of abdominal distention in the aged includes fluid (ascites), flatus, feces (impaction), forms of neoplasia, and fat. There is an increased prevalence of fecal impaction, which may present as abdominal pain and vomiting, or as nonspecific decline. Feces are frequently palpable in the sigmoid colon, where they simulate a mass, and occasionally elsewhere. Two maneuvers are called for. First, palpation is repeated with the abdominal muscles contracted, by having the patient try to sit up or be pulled up while one palpates. If the mass is unchanged, unless the muscles are paper-thin it must lie within the abdominal wall, not inside the peritoneum. Second, the colon is evacuated. If the "mass" disappears, it was feces, and the patient is spared diagnostic testing for cancer.

Fecal impaction can coexist with a normal rectal examination if all the stool lies in the sigmoid and above. Hard stool in the rectal vault proves that a disorder of rectal emptying is present, since feces in the ampulla stimulate defecation in the presence of intact mechanisms.

The anorectal area is also examined for fissures, hemorrhoids, and when at issue, sphincter tone. Testing sacral-dermatome sensation and the anal "wink" reflex are parts of the neurologic workup for incontinence. The wink consists of visible contraction and puckering when the medial thigh is stroked with a cotton swab.

Fecal occult-blood testing is often part of the physical examination. False-negative tests frequently occur even with cancer, so a negative test cannot be interpreted as conclusively reassuring. Ingestion of vitamin C is one specific cause of false-negative results. False positive results may follow ingestion of lightly cooked meat. Data on whether oral iron supplements produce false-positive tests *in vivo* as they do *in vitro* are conflicting.

Estimation of prostate size by digital examination can disclose enlargement, but cannot exclude it: if the prostatic urethra is obstructed because of focal hyperplasia of the median bar producing unequivocal prostatism, there may be no abnormality in the accessible posterior portion. Although digital examination may discover prostate cancer at an early stage, many cases present with advanced disease despite a negative competent examination a year beforehand. While any hard prostatic nodule requires workup, three-fourths turn out not to be cancers but foci of nodular hyperplasia, granulomatous inflammation, prostatic infarction, or calcinosis. A prostate that is tender, swollen, and diffusely indurated may harbor acute prostatitis.

Retraction of the foreskin remains valuable for the demonstration of lesions. Early penile cancer produces a reddening that does not regress after therapy for presumptive balanoposthitis.

Testes sometimes show reduced size in extreme age. For intrascrotal masses, lymphoma replaces testicular germ-cell neoplasm as the leading malignancy in old age.

Examination of the female external genitalia can be satisfactorily completed in bed or Geri-chair, free of the excuse applied to speculum examination, that assuming lithotomy position represents undue strain on a frail old woman. Vulvar dystrophies are recognized by discolored patches on the labia, usually with some asymmetric shrinkage of labial supporting tissue. Atrophic vulvovaginitis produces a yellow-white to reddish mucosa that is recognizably friable, and often contains telangiectases or petechiae. A major distinction is from recurrent candidal infection, which is overdiagnosed. Vulvar cancer is recognized by persistent focal discoloration, with or without a mass or nonhealing ulcer.

Bimanual examination can proceed without speculum examination or Papanicolau smear. The latter two are desirable, since cervical cancer in the aged is rather common, and its mortality largely preventable by early cytologic diagnosis. However, partial assessment is better than none at all, and one ought not omit inspection and palpation simply because the full procedure cannot be completed. For the woman with a contracted introitus or vaginal synechiae (mucosal adhesions), monodigital vaginal palpation is usually feasible, particularly if done gently, with continuous calm talk, and with a chaperone to hold the hand and comfort the patient. The uterus is usually impalpable in the aged, and any adnexal tissue that one can feel suggests a mass and calls for repeat palpation postevacuation, to be followed by ultrasound imaging or referral if persistent.

Musculoskeletal system.—Contractures are noted and muscle strength tested briefly. Symptomatic joints are examined for swelling, deformity, tenderness, crepitus, warmth, and redness. Both the active and the passive range of motion are tested if symptoms exist.

Brief screening tests for upper limb function—joint, muscle, nerve, and integration—related to the upper arm are "Touch the back of your head with both hands"; for the hands and forearms, "Pick up this spoon."[2]

Scoliosis deserves notice. So does excess kyphosis resulting from multiple thoracolumbar osteoporotic compression fractures. Either may explain back pain and part or all of respiratory compromise. Local spinous process tenderness may occur in diverse settings. Except in the patient who has global hyperalgesia, it is worth evaluating further.

A stance resembling that of a nonhuman primate has been described in lumbar spinal stenosis.[19] This may minimize pressure on the pathologic areas, and so diminish pain. It can be voluntarily overcome but offers valuable insight if recognized.

A functional assessment of gait and mobility consists of having the patient rise from a chair, walk with any assist device, such as a rolling walker, that she habitually employs, make a complete 180° turn, raise each foot completely off the floor in turn, return, and sit again. This sequence correlates well with functional impairment in the home, better than standard neuromuscular examination.[20] An even simpler sequence is, "Rise from your chair, walk three meters (ten feet), return, and sit down."[2] One observes every aspect of these sequences closely.

Hands and feet.—Onychomycosis, fungal infection of the toenail, causes thickening and yellowing. Associated infection of the interdigital space may produce cracking and loss of the epithelial barrier, a portal of entry for cellulitis that can be identified on physical examination. Ischemia can mimic onychia by producing a hyperkeratotic, thickened, dystrophic nail.

Plantar ulcers are commonest in diabetes, but may occur in any person with neuropathy, arterial insufficiency, or both. Sometimes neuropathy renders these lesions painless, and unless the patient has noticed the sore or a discharge on socks or stockings, discovery may await physical examination. The need to diagnose diabetic foot ulcers early is urgent: recent work shows an extraordinarily high prevalence of osteomyelitis, not only in exposed bone, but even beneath shallow lesions.[21] Such

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

osteomyelitis is frequently a precursor to amputation, but diagnostic techniques and potent therapy can refine assessment and influence natural history favorably.

Neurological and mental status examinations.—If there is a question about slight hemiparesis, the digiti quinti sign can be helpful: with eyes shut and hands outstretched, the fifth finger on the affected side tends to drift outward. If this occurs bilaterally, the fingers can be gently squeezed into apposition, or the patient can be told, "Now put your fingers together and stretch out your arms". Persistent abduction may raise the question of cervical spinal cord disease; to confirm the latter, one looks for isolated inability to maintain the fifth finger adducted for more than 30 seconds, despite intact abduction and opposition to the thumb;[24] with progression, the ring and middle fingers can develop the same deficit, and the number of grip-release cycles that can be performed in 10 seconds can become markedly attenuated.

Stiffness may reflect several problems, including Parkinsonism, side effects of medication, and cognitive dysfunction. Cogwheel rigidity can be enhanced by having the patient draw a circle in the air continuously with the hand contralateral to the side being tested. There are no data on the frequency of false-positive signs with this maneuver.

Three causes of abnormal involuntary movements in the aged are essential tremor, Parkinson's disease, and extrapyramidal side effects of neuroleptic medication. Parkinsonian tremor occurs at rest, disappears during sleep, and may reappear during the rapid-eye-movement (REM) phase of sleep. It is most pronounced distally in the extremities, beginning asymmetrically in the fingers and thumb. One study suggests that non-essential upper limb tremor can be enhanced by having the hand held near the subject's face. A scale for rapid, standardized bedside assessment of abnormal involuntary movements was developed in relation to extrapyramidal side effects, but is applicable regardless of etiology.[23] The development of myoclonus on being suddenly startled has been considered a hallmark of Creutzfeldt-Jakob disease. However, this feature is found in 5% of patients with the far commoner Alzheimer's disease.[24]

While vibratory sensation in the legs decreases with aging, position sensation does not. It is reasonable to test position sense exclusively, and to consider disease of the posterior columns if it is defective.

Ankle jerks can be lost due to age alone, but should not be lost asymmetrically. Reinforcement with the Jendrassik maneuver or an equivalent is essential if the unreinforced reflex cannot be elicited. Many elders are unable to kneel on a chair, the ideal position for this test, but most can sit with the foot flat against the floor to assist relaxation of the musculature about the ankle joint and to produce a trace of passive dorsiflexion that improves the test. The plantar reflex is tested in the conventional way. The alternative Oppenheim method of forceful stroking down the anterior shin can produce ulceration and hematoma formation with thin fragile skin and venous insufficiency.

Gait difficulties, particularly of acute onset, can occur as nonspecific "weak link" responses to major illness of unrelated systems. In normal pressure hydrocephalus, initiation of gait is difficult and step height is reduced, resulting in a shuffling walk whereby the feet appear to be stuck to the floor. Gait abnormalities may be present without the two other elements of the triad, urinary incontinence and dementia.

Spondylotic cervical myelopathy can cause a spastic, shuffling gait disturbance that may interfere with complex processes (eg, climbing stairs) before it affects walking on a level surface. Neck flexion and extension impairment, or reproduction of symptoms, are not reliable guides to the presence of this condition. Muscle stretch reflexes are hyperactive or clonic below the level of compression, muscle tone is increased, and adduction of the fifth finger is commonly defective.

Signs of frontal lobe dysfunction such as grasp and snout reflexes occur in some normal old people. Unilateral signs appear to be more useful than symmetric ones.

The diagnostic interview is notoriously insensitive in detecting brain dysfunction as long as the subject displays no agitation and behaves in a socially appropriate manner. The mental status portion of the physical examination serves brain assessment more effectively. A screening procedure is to name three objects, such as pencil, truck, and book, and then to have the patient repeat them immediately and again one minute later.[2] A more sophisticated test is the standardized mini-mental status examination (SMMSE). This series of questions and tasks is simple to learn and administer.[25] The SMMSE underestimates cognitive function in persons with major sensory or motor problems, non-English-speakers, and those with limited formal education. It underestimates *deficits* in highly educated and verbally fluent subjects.

The Draw-a-Clock test is used for rapid assessment of nonverbal cognitive function, and consists of handing the subject a paper with a circle on it, and giving the simple directions, "First, draw a clock with all the numbers on it. Second, put hands on the clock to make it read 2:45". Criteria for scoring and interpretation have been published.[26]

Delirium is an acute, reversible brain dysfunction associated with almost any physical ailment.[12] Its detection carries enormous prognostic and therapeutic implications. Delirium evaluation encompasses more than the mental status examination, but begins with this assessment, as does the workup of those most challenging disorders, the aphasias. Discussion of these critical areas exceeds the scale of the present review.

Conclusion

Physical examination of old people, be they well or ill, requires, taxes, and sometimes exceeds the resourcefulness and creativity of the physician. So does obtaining their history. These difficulties may render the care of the old frustrating, but the delight experienced from success in eliciting the subtle diagnosis and the comprehensive data base is immense. It is hoped that the preceding account, which covers only some highlights and has no pretensions to being comprehensive or all-inclusive, has also conveyed some of the pleasure to be had in the process of accumulating information on challenging elderly patients.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

REFERENCES

1. Sackett DL: A primer on the precision and accuracy of the clinical examination. *JAMA* 1992; 267:2638-44.
2. Lachs MS, Feinstein AR, Cooney LM Jr., et al: A simple procedure for general screening for functional disability in elderly patients. *Ann Intern Med* 1990; 112:699-706.
3. Lagaay AM, van der Meij JC, Hijmans W: Validation of medical history taking as part of a population based survey in subjects aged 85 and over. *Brit Med J* 1992; 304:1091-2.
4. Morley JE: Why do physicians fail to recognize and treat malnutrition in older persons? *J Am Geriatric Soc* 1991; 39:1139-40.
5. Gambert SR: Nutritional assessment in the elderly. *Wisconsin Med J* 1982; 81:18-21.
6. Lichtenstein MJ, Schaffner W: Assessing activities of daily living. *Hosp Practice* (May 30) 1985; 20:8-9.
7. Meissner HH, Franklin C: Extreme hypercapnia in a fully alert patient. *Chest* 1992; 102:1298-9.
8. Castle SC, Norman DC, Yeh M, Miller D, Yoshikawa TT: Fever response in elderly nursing home patients: Are the older truly colder? *J Am Geriatr Soc* 1991; 39:853-7.
9. Sykes D, Dewar R, Mohanaruban K, et al: Measuring blood pressure in the elderly: Does atrial fibrillation increase observer variability? *Brit Med J* 1990; 300:162-3.
10. Curran RE: Palpation of the superficial temporal artery in normal persons. *Arch Ophthalmol* 1986; 104:1756.
11. Hunder G, Bloch DA, Michel BA, et al: The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis and Rheum* 1990; 33:1122-8.
12. Lipowski ZJ: Delirium in the elderly patient. *N Engl J Med* 1989; 320:578-82.
13. Mashberg A, Feldman LJ: Clinical criteria for identifying early oral and oropharyngeal carcinoma: Erythroplasia revisited. *Am J Surgery* 1988; 156:273-5.
14. Sofferman RA: Lingual infarction in cranial arteritis. *JAMA* 1980; 243:2422-3.
15. Puxty JAH, Fox RA, Horan MA: The frequency of physical signs usually attributed to meningeal irritation in elderly patients. *J Am Geriatr Soc* 1983; 31:590-2.
16. Ruiswyk JV, Noble H, Sigmann P: The natural history of carotid bruits in elderly persons. *Ann Intern Med* 1990; 112:340-3.
17. Ballard DJ: Selective screening for abdominal aortic aneurysms with physical examination and ultrasound. *Arch Intern Med* 1989; 149:1463-4.
18. Christensen JH, Freundlich M, Jacobsen BA, Falstie-Jensen N: Clinical relevance of pedal pulse palpation in patients suspected of peripheral arterial insufficiency. *J Intern Med* 1989; 226:95-9.
19. Simkin PA: Simian stance: A sign of spinal stenosis. *Lancet* 1982; 2:652-3.
20. Tinetti ME, Ginter SF: Identifying mobility dysfunctions in elderly patients: Standard neuromuscular examination or direct assessment? *JAMA* 1988; 259:1190-3.
21. Newman LG, Waller J, Palestro CJ, et al: Unsuspected osteomyelitis in diabetic foot ulcers: Diagnosis and monitoring by leukocyte scanning with indium 111 oxyquinoline. *JAMA* 1991; 266:1246-51.
22. Ono K, Ebara S, Fuji T, Yonenobu K, Fujiwara K, Yamashita K: Myelopathy hand: New clinical signs of cervical cord damage. *J Bone Joint Surg* [Br.] 1987; 69-B:215-9.
23. Munetz MR, Benjamin S: How to examine patients using the abnormal involuntary movement scale. *Hosp Comm Psych* 1988; 39:1172-7.
24. Burns A, Jacoby R, Levy R: Neurological signs in Alzheimer's disease. *Age and Ageing* 1991; 20:45-51.
25. Molloy DW, Alemayehu E, Roberts R: Reliability of a standardized mini-mental state examination compared with the traditional mini-mental state examination. *Am J Psychiatr* 1991; 148:102-5.
26. Wolf-Klein GP, Silverstone FA, Levy AP, Brod MS, Breuer SCJ: Screening for Alzheimer's disease by clock drawing. *J Am Geriatr Soc* 1989; 37:730-4.

What Matters Most!

PETER J. DECKERS, M.D.

THE current status of general surgical practice is, I believe, best summarized by two of the most famous quotations in English literature. They are found in the same book, one at its beginning, "It was the best of times, it was the worst of times, it was a season of light, it was a season of darkness," and the other at its end, "It is a far, far better thing I do than I have ever done." The source of these quotes is, of course, *A Tale of Two Cities*, a novel about the French Revolution by Charles Dickens. These familiar words come to mind as I reflect on what has occurred to us during the past 10 years and ponder what lies ahead.

A staggering revolution has occurred in health care during the past two decades, which will continue and increase in intensity in the next 10 years. Some among you are knowledgeable about it, and some care deeply about its implications for their patients and society itself. However, many seem strangely indifferent to it, being either unable, or not caring enough, to adjust, be flexible, adapt to change, or participate constructively in it. Our entire training was designed, perhaps appropriately so at the time, to make us consistent, methodical, compulsive, narrowly interested, constant, and careful in our thinking and actions. Unfortunately, this is probably responsible for our present attitude. Also, our training forces us to grow up, and to grow up quickly. We generally handle life and death effectively and efficiently; in this, we are the envy of our peers. We can also do "wondrous things" today. We can cause some of the blind to see, some of the lame to walk, some of the deaf to hear, and some of the dumb to speak. Through the miracle of immunosuppression, advanced organ transplantation is a reality, and some of the almost-dead can be restored to very useful life. We have an expanding number of effective anticancer agents. Especially significant advances have been made in childhood malignancies. Many previously incurable cancers are now curable. CT scanning and magnetic resonance imaging are revolutionizing our diagnostic and potentially therapeutic abilities. Antibodies generated from so-called "immortal cells" may be specifically targeted either alone or in combination with lethal toxins, drugs, or radioactive materials against tumor cells. We are rapidly learning to identify some of the genes responsible for cancer. We may be able to modify their phenotypic expression, prevent it, or even correct through genetic engineering base pair sequences in DNA at specific gene loci, thereby eliminating disease. We have learned to harness light in a splendid device known as the laser. With it, we can vaporize cancer, open blocked arteries, and seal leaking blood vessels, all the while leaving tissue unaffected only a few cell widths away. Other new technologies, either in our hands (laparoscopy) or in the hands of the interventional radiologist, cardiologist, or gastroenterologist, together with a better understanding of pathophysiology (especially tumor growth and metastasis) have eliminated or threatened with extinction such previously common general surgical procedures as gastrectomy for duodenal ulcer, sympathectomy in cardiovascular disease, staging laparotomy in Hodgkin's disease and, perhaps soon, axillary dissection in breast cancer. If you will not accept the term "revolution" to describe the current changes in health care and medical education, at least let us agree that we are in an era of profound transition. Indeed, the changes are so real that we can scarcely remember the primitive state of medical science practiced as recently as 40 years ago. Medicines now as mundane and commonplace as vaccines, antibiotics, and biologic response modifiers were not available to our predecessors during World War II. For generations, we analyzed disease but rarely treated it. Medicine was,

---

PETER J. DECKERS, M.D., Murray-Heilig Professor and Chairman, Department of Surgery, Interim Dean, University of Connecticut School of Medicine, Farmington.

The above address was delivered at the Annual Meeting of the Connecticut Society of American Board Surgeons in December 1992.

as Lewis Thomas remarked, "in real life a profoundly ignorant occupation."

We have made staggering advances in the past three or four decades. There is no question that we now do, in fact, treat disease and treat it well. We practice in an exciting, stimulating "brave new world." But at what a price! How did this electrifying pace of medical discovery occur? To finance the research necessary, millions of dollars of federal aid were required. Medical school faculties and facilities grew dramatically in the past 30 years—whether this was necessary or not. Spurred on by our "moonwalk" mentality, we accepted, for one, the challenge that cancer could be cured in a decade—as long as enough money was spent! Research efforts, many lacking discrimination and objective peer review, surged forward. Teaching hospitals became temples with equipment of immense sophistication and complexity. An era of specialization in medicine necessarily developed. After all, given the sophistication and complexity of our new science, our medical confreres would have us believe, for instance, that not everyone could be expected to administer chemotherapeutic agents, despite the fact that most are not much more complicated than antibiotics from a pharmacologic perspective.

Doctors today are not doctors in the sense that our fathers or mothers understood them. They are not easy to get on the phone. They do not make house calls. Visits are "by appointment only." Physicians have become nephrologists, cardiologists, pulmonologists, etc. Radiologists are now interventional, diagnostic, or therapeutic. Surgeons are, unfortunately no longer generalists, but are instead oncologists, traumatologists, burn specialists, or intensivists, or they have received "special certification" in colorectal, critical care, endocrine, hand, or vascular procedures. Issues of turf are commonplace, and credentialling today for inclusion on hospital staffs is more often political than objective. Delineation of surgical privileges within specific departments of a given hospital often borders on marketing on the one hand or restraint of trade on the other.

Advanced technology and a better understanding of pathophysiology breed specialization. I am sure the majority of this audience has settled on a particular specialty in surgery out of personal interest, either willingly or out of necessity. In and of itself, this is not bad because such technology, knowledge, and specialization are certainly responsible for many of the real advances in medicine in the last two decades. However, can this specialization, this proliferation in technology, this unbridled competition and the unchecked use of diagnostic and therapeutic resources continue? What are the fiscal consequences? As a nation, we already spend 880 billion dollars (14% GNP) each year on health care. This figure is expected to reach a trillion dollars (17% GNP) by 1995. Where will the money come from? The answer is clear—from other government entitlements! This has the custodians of these entitlements deeply worried. Also, what are the solutions to the ethical dilemmas created? For instance, who will have access to such detailed and expensive care? How much will be provided? Already, 35 to 40 million Americans are not covered by health insurance—and 80% of them are employed! An additional 20 to 25 million Americans are underinsured. How will we decide which technology has reasonable, immediate applicability? What group (or groups) will govern these decisions? Will our new president have his way? Will a National Health Care Board be created to formulate a comprehensive benefits package for all of our citizens? If created, will this really "provide full protection in case of illness," as promised? Will patients really be able to choose where they receive their treatment? Who will be the boss—the patient (consumer), the physician (provider) or a Managed Care Plan, or Uncle Sam? Most importantly, who will be the patient's advocate?

These questions of high cost and access are the issues with which we must concern ourselves in the next decade. These will matter! They are the major problems of medicine. Politicians and business coalitions cannot, and should not, solve our problems. There must be clear, rational physician input. Our answers to these questions will make a difference. In the recent past, the federal government, acting appropriately through its Medicare and Medicaid programs to assure health care to the elderly and the poor, did so at a cost that has come to exceed 90 billion dollars a year. The ready availability of these services led to their use—needed or not. We found that people like to see their doctors, whether they need to or not. Unfortunately, we also found that some doctors also like to see patients—sometimes whether they need to or not. To satisfy the expanded use of medical services to these programs, new hospitals were rapidly constructed. More doctors were trained—again, whether they were needed or not. We soon began to realize that the stimulus of government-financed, cost-reimbursed programs might be working too well. We became, and remain, an over-bedded, over-doctored nation. Tragically, despite all the incentives, there is still a maldistribution of physicians. Some prophets, still today perhaps whistling in the wilderness, have even dared to come full circle, suggesting that too many doctors are specialists and far too few are generalists—by which, by the way, is meant pediatricians as well as family medicine and general internal medicine primary care doctors, not general surgeons. Moreover, it has been more than obliquely or subtly stated that "primary care physicians may identify patients who are NOT appropriate candidates for a procedure more effectively than the

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

specialist who performs the procedure."

The worst may be yet to come. Incentives to restrain expenditures represents a relatively new idea. Government rules now exist to encourage the rapid development of supposedly less expensive, prepaid HMOs, to restrict new hospital construction, to fix the reimbursement for most hospital medical services through DRGs, and to fix and decrease the cost of physician's fees through RBRVS. As most of you realize, this is already occurring. Between 1981 and 1986, the growth in Medicare physician expenditures was 15%; between 1986 and 1991, it was 11%. Surgical services, specifically, took the biggest hit, increasing only 6%. Parenthetically, in many discussions I have heard that the only way to curb health care costs would be to pay hospitals on a disease-specific basis for not only the hospitals' costs, but for the physicians' costs as well—one fee for the total service. The hospital would then decide what percent of the total dollars it would allot to the physician of record. A chilling concept, you say. No way, others would say. Maybe! Remember that other such previously considered impossible notions now exist. Perhaps more immediate is the concept of one global fee for the total service rendered to a surgical patient (including preop, in-hospital, and postop considerations) for a specific illness. Also to be considered is, will the next president succeed in setting national and state health care budgets to be applied to managed care insurers, nonmanaged care insurers, and the Medicare programs, as he proposes? Indeed, we have come full circle. Unbridled expenditures in medicine will not guarantee quality health care to all of our citizens. Incentives exist now to restrain costs, and more will he developed. The challenge is, can we do this without diluting the quality of care we have achieved? Can we still allow enough room for creative individualism in basic and clinical research, the so-called right stuff from which real advances occur? This then is the environment in which we find ourselves. What can we do?

We must remember that we are, first and foremost, doctors of medicine. Our commitment is to people. The art of healing serves directly and principally the person, not the State. We have a profound moral obligation not only to our patients, but also to their families. We must completely enter the lives of the sick and dying. We must reach out not only to the insured, but to the outcasts, the deprived, the despised, the downtrodden, the poor, and to all under-represented minorities. We must not fix our system economically by reestablishing two health care systems: one for the affluent, one for the poor. As physicians, we have a special ministry. If we are admired at all, it should be because our "healing power," if you will, has as its root compassion for all who suffer illness. THIS IS WHAT MATTERS MOST—our patients and our relationship to them.

What then are some of the immediate issues we must face?

1. *We must help to define the appropriate relationship between hospital management and medical staff.* Today, payments to hospitals for inpatient services are made on a diagnosis-specific basis, in sharp contrast to cost-based retrospective reimbursements. Since physicians control 60-70% of health care expenditures, hospital managers must attempt to convince the medical staff to adhere to cost-effective practice patterns. We fail miserably in this regard. Therefore, great potential exists for a volatile relationship between a medical staff devoted to good care (until recently, regardless of cost) and administration.

We are aware that the physician glut has caused a stabilization of, or a reduction in, our incomes. No longer can we become wealthy in medicine. Similarly, the financial stability of hospitals may decrease due to a shrinking demand for inpatient care. As we have already seen, both groups (physicians and hospitals) will attempt to diversify by opening surgical centers or satellite primary care clinics, and expanding outpatient or office services. Tension between the two groups is bound to increase. Administration must attempt to convince you to violate the manner in which you have been trained and to adopt the less is better philosophy. Sophisticated information systems have been set up regionally and nationally to monitor or, if you will, spy on physician behavior. Mechanisms must he established to reeducate or, if necessary, dismiss cost-inefficient physicians.

Definition of cost-effective standards of care is where we must become involved. Physicians cannot sit back on this one as they have done on so many issues in the past, allowing politicians, administrators, actuaries, or businessmen to determine the quality and quantity of care our citizens receive. None of these groups understands excellence in medicine. THIS IS WHAT MATTERS MOST! As physicians, we can and must establish rigid, effective, cost-efficient protocols, standards of care, or clinical pathways—call them what you will. Please realize that protocol care need not be "cookbook" medicine. It should not be feared. In fact, adherence to well-organized, collaborative, carefully thought-out protocols cannot only be cost-effective, but they may ensure a more consistent level of excellent and safe care. This is in sharp contradistinction to the variability in quality and the great variation in volume of services used—which all of us know exist now from physician to physician, hospital to hospital, state to state, and region to region in the United States. This variability has been self-evident to physicians for a long time, but it has been almost completely ignored. If you would doubt its existence, there are now many studies similar to the Stanford study of the early 1980s, which reviewed the mortality and morbidity rates for 8,593 patients at 17 California hospitals. These researchers found the recovery rate to be nearly 250% better at the hospital rated best by the study compared to the one at the bottom of the list. Isn't this degree of variation large enough to constitute an important health care problem? Can we as physicians continue to ignore the fact that peers are not always equals?

We must acknowledge that our so-called Peer Review and Quality Assurance (QA) committees are not doing their jobs! Moreover, effective QA needs to be more than just a mere counting of morbidity and mortality. Additionally, good QA requires analysis of judgment and long-term outcome assessment or functional analysis from the patients perspective. Further, it should be dynamic and interactive with the physician at the time care is being delivered. If we can agree, as we must, that competency varies amongst physicians, then protocols for clinical care may be one way to dilute this inadequacy, thereby offering all patients an equal chance for good quality care. The protocol studies of the National Surgical Adjuvant Project for Breast and Bowel Cancers are an outstanding example of how collaborative efforts by multiple physicians can painlessly lead to development of excellent protocols for uniform preoperative evaluation, intraoperative care, and postoperative follow-up, which today are considered "state of the science" care across the country. More than anything else, they have advanced our knowledge about breast cancer by asking and answering specific important biologic questions of major concern. Data from therapeutic interventions in Stage I and Stage II breast cancer have even led to the development of reasonable strategies to prevent the disease in high-risk women. The NSABP protocols represent the reference point against which all other physicians can compare their results.

2. *We must be faithful to the hospital of our choice. Multiple hospital affiliations will not do.* We can no longer spread ourselves too thin. Choose your hospital well, and then consider it your home, part of your extended family. Remember, it cannot succeed without our help. Simple, passive fidelity, so characteristic of so many physicians, is not enough. Our fidelity must be creative and contributive. We must actively seek out the opportunity to serve on Credentials Committees, Medical Records Committees, Medical Staff Councils, Executive Committees, and Institutional Review Boards, as well as Peer Review Quality Assurance, and Utilization Review Committees. THIS IS WHAT MATTERS MOST! These committees will have considerably more influence in the future. They will influence development of protocols or clinical pathways and they may even establish the basis for medical payment in time. Be active, be involved, make the sacrifice, hurt a little, give of yourself without expecting too much in return.

3. *We must help answer the question, "Who will play God?"* In other words, what is your responsibility to hopelessly ill patients? Today, our skills and technology can prevent death. We can prolong or sustain life a few months or a year, but at what a price—emotional and fiscal! Just because we can extend life, are we obliged to do this? Must we die with electronic gadgetry and intravenous solutions running? We must not only ask the question, when do we use our medical knowledge and techniques? We must now ask, when is it appropriate and humane to forgo life-prolonging therapy? When do we NOT act? Courage will he required in this issue. Many hospitals for fear of legal reprisals still lack clear procedures for observing and recording the wishes of dying patients. Often, the decision of whether or not to call in the resuscitation team is left to some young nurse or resident on duty in the middle of the night. This is not their responsibility. It is yours and mine, and only ours. The ability to make such decisions after intimate communication with our patients and their families is at the essence of what it means to be worthy of the term "my doctor." THIS IS WHAT MATTERS MOST!

4. *The staggering advances in transplant surgery, of which therapeutic molecular biology such as gene therapy may be the ultimate expression, will continue to have a profound impact on us and our hospitals.* These procedures do prolong useful life and also offer significant relief from physical, emotional, and fiscal suffering. Can the demand for donor organs be allowed to continuously exceed the supply? Can one's personal prejudice or religious viewpoint be supported with no concern for the possible help to the living that the dead body can provide? Perhaps the highest reverence for the dead may be the mandatory use of their organs for transplantation. Today we have the ability to act to an unprecedented degree. In Connecticut, we have two excellent transplant programs, one at Yale and one at Hartford Hospital, both of which are dramatically underutilized. We can do much good if we are willing to act lovingly, compassionately, and decisively. It is now comfortable for us to aggressively pursue intensive care medicine. We will have matured as physicians when we learn to throw off the enslaving shackles of our technology and learn, for instance, to be similarly aggressive in seeking transplantable organs. No one can help us with this. It is our responsibility. We cannot avoid it. THIS IS WHAT MATTERS MOST!

There are many other issues. We cannot cover all of them here today. In conclusion, therefore, I hope these words have convinced you that the first characteristic of an excellent physician must be love of humanity and respect for life. We must enhance the lives of those we care for. We must be patient advocates. We must relieve suffering and make available to all as far as is possible the opportunity for a full human life. Quality patient care at its highest level is an amalgam of virtues, including judgment developed from experience, intellectual curiosity stimulated through honest, careful analysis of basic and clinical scientific investigations, and moral responsibility derived from the human intimacy of our profession. We are privileged people by virtue of our training, our title, and our responsibilities. Much is required, even demanded, of us. What we do is crucial and important. As protectors of health, we are, as Virchow said over 100 years ago, protectors of humanity. This is our calling. This is our commitment. As physicians none of us really has a job. We have a unique privilege and opportunity.

Our profession is the most profound and the most noble. It has been peered at under a microscope, dissected and reassembled, mounted for public display, examined psychologically, economically, sociologically, and philosophically, and trivialized by authors and serializations with a sentimental bent. But it endures, and it will endure always. We must help in this battle. Resolution of the issues we face will not be easy. We must not move away and hide in our science, our technology, our specialization. We must be involved, be committed. Remember, our patients matter, we matter, and our profession matters!

CALL FOR PAPERS

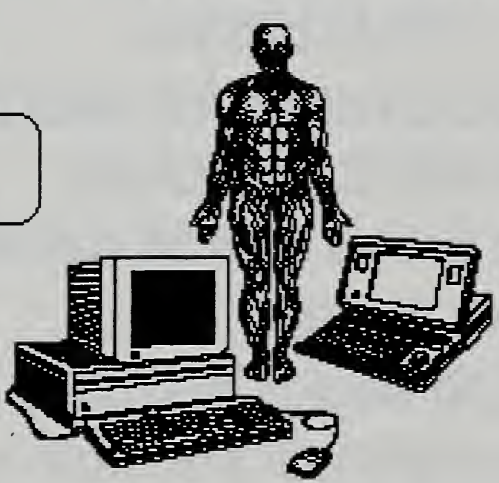
Members of the Connecticut State Medical Society reading papers before other organizations are requested to submit their papers to the JOURNAL for consideration by the Board of Editors for publication. Authors preparing manuscripts for submission to *Connecticut Medicine* should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office (also printed in *Connecticut Medccine*). Adherence to the instructions will prevent delays both in acceptance and in publication. A copy of the manuscript on floppy disk (3-1/2" or 5-1/4") should be submitted along with the hardcopy.

Please send them to:
Robert U. Massey, M.D.
Connecticut Medicine
160 St. Ronan Street
New Haven, CT 06511

GRATEFUL MED

$29.95

SOFTWARE
PASSWORD
$40. CREDIT



AVLINE
CANCERLIT
CATLINE(books/jrns)
CHEMLINE
DIRLINE(organizations)
HEALTH(non-clinical)
MEDLINE(biomedical)
PDQ(cancer)
SDILINE(current-month)
TOXLINE
TOXLIT
TOXNET

FREE INSTRUCTIONAL WORKSHOPS

NEW FEATURE :
USERS CAN PLACE AN ONLINE ORDER TO RECEIVE FULL-TEXT ARTICLES FROM PARTICIPATING HOSPITAL OR ACADEMIC LIBRARIES

For futher information contact:
National Network of Libraries of Medicine
New England, Region 8
University of Connecticut Health Center Library
263 Farmington Avenue
Farmington, CT 06030-5370
TEL: 800 338 RMLS or 203 679 4500

INQUIRE TODAY.

ENDORSED BY
THE CONNECTICUT STATE MEDICAL SOCIETY
—OFFERING—

- Hospital, Surgical and Major Medical Insurance
- Term Life Insurance
- Employee Benefit Package

—FEATURING—

- OFFICE OVERHEAD AND DISABILITY INSURANCE PACKAGE
  [Rates based on a 30 day waiting period for ages 30-39]
  $1500.00 weekly disability indemnity
  semi-annual premium is $528.15*
  $20,000.00 monthly overhead indemnity
  semi-annual premium is $561.00

* Optional Residual and COLA benefits available

AYR INSURANCE AGENCY

105 SANFORD STREET HAMDEN, CT 06514 288-4302

The Connecticut State Medical Society and Professional Liability Insurance 1971 to 1983

W. RAYMOND JAMES, M.D.

THIS is the second of three articles on the history of medical malpractice and CSMS. The period with which this article is concerned began after 40 years of unwarranted public and professional complacency. During this period CSMS's only recorded concerns were with equitable rates and availability of insurance. Neither was fully achieved. Premiums with the Aetna Life Insurance Company do seem to have been remarkably low.

By 1970 the increasing complexity of medicine and of the physician-patient relationship was changing professional liability insurance. From a necessary but incidental, and largely ignored, part of medical practice it became one of the primary concerns of all physicians and the major expense in many practices. Broadening legal definitions of incompetence, negligence, breach of contract, assault, and others, and new concepts of liability, such as loss of consortium, wrongful life, cost of child rearing, and an ever-growing list had rapidly increased the incidence of lawsuits. Unusually high punitive damages, unreal values placed on pain and suffering, and enormous assessments of damages had greatly inflated losses. The rate of premium increase during this period far exceeded that of inflation, with little prospect of slowing in the near future. Standardized premiums were replaced by class premiums dependent on physician specialty and insurance exposure.

In 1970, following the recommendation of the Committee on Insurance, the Society appointed another ad hoc committee, chaired by Dr. Alfred Fabro, to study and report on this increasingly complex and expensive field. In April 1971 the House of Delegates of the Society, following the committee's recommendation, approved an agreement with the Aetna Insurance Company. In exchange for endorsement, the insurance company agreed to make policies available to all Society members for a five-year period (renewed in 1976 for another five years). Provisions were made for peer review prior to cancellation, for claims review by physician panels, and for annual reports. Aetna would support educational programs with the Society. Premiums during the first years, ranging from $445 for Class I to $2,549 for Class 5, were among the lowest in the nation. By August 1980, in spite of yearly increases in premiums, Aetna's conservative estimates of their accumulated underwriting losses, during the agreement period, came to approximately 12 million dollars.[1] Later revised estimates exceeded 30 million dollars. This "loss" did not take into account Aetna's gain from investment income.*

---

*The average length of time from event to settlement is about 5-1/2 years. Premiums set aside to cover anticipated losses are invested during this period. Investment income each year at the then high interest rates amounted to as much or more than annual premium income.

W. RAYMOND JAMES, M.D., is a member of the CSMS Council and alternate delegate to the AMA. Dr. James is past president of CSMS and has been in the private practice of medicine in Essex, Connecticut, since 1948.

A continual source of friction between Aetna and the Society's committee was the unavailability of investment income data to the Society. Later it became evident that the Aetna representatives did not have these figures. The generally favorable experience with Aetna during that decade was marred also by several major changes that were made without consultation with the Society. The first was the abolition of the right of the physician to determine whether his case would be settled out of court or be tried. (The settling of doubtful or nonmeritorious cases, even at low rates, is thought to encourage more suits). Physician-owned companies, like the defense committees from the early 20th century, have settled only in cases of obvious liability and defended other cases aggressively. Aetna's second arbitrary action, taken without consultation with the state Society or with the Hartford County Medical Association (HCMA), was the development of a pilot plan with Hartford Hospital and the Hartford Hospital medical staff. The plan offered lower premiums dependent on successful loss reduction through concerted hospital and medical staff quality control and educational efforts. It was essentially the plan being considered by Aetna and the Society that year.

By 1975 the majority of insurance companies throughout the United States, faced with progressively increasing losses and the uncertainties of the "long tail,"* discontinued professional liability insurance on short notice. The Society's arrangement with Aetna, and the generally favorable tort law in Connecticut, made it possible for Aetna and the CSMS to weather this tumultuous year without significant changes in rates or in the original agreement. Indeed, the state Society received a considerable boost in membership of over 800 physicians who suddenly found themselves without other sources for liability insurance.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

The Society's major action during this crisis was to approach the legislature with an extensive list of proposed tort reforms and to appropriate 100,000 dollars for expenses thereby incurred. In response, the legislature did appoint a commission to study professional liability. The only other legislative change guaranteed peer review immunity making possible greater physician participation in claims management and risk prevention programs and in the identification of high-risk physicians. The state Society continues to have a strong legislative program with lobbyists assisting the staff and an effective legislative committee. During the 1971 to 1983 period no tort legislation opposed by the Society was passed although much threatening legislation was introduced. (Efforts to lengthen the time in the statute of limitations still must be resisted every year!) Physicians were not the only group threatened by the increasing litigation in this century. Many others, from individuals to industry, municipalities, and even nursery schools, were damaged by many adverse court decisions in liability cases. There was a marked decrease in available liability insurance as well as great increases in the cost of such insurance. Previously alone in attempts at tort reform, physicians now found industry, municipalities, and even churches supporting reform, and some significant legislative changes were made in the 1980s. Some of these were reversed shortly after passage by a very active trial lawyer's organization (note that many legislators are lawyers).

Although Aetna had increased premiums steadily throughout the first eight years, these increases were usually in line with inflation and produced little reaction in the Society. But in 1979 Aetna announced a 25% premium rate increase. The response of the Society, through a new Professional Liability Committee, chaired by Dr. Gioacchino S. Parrella, was to appoint subcommittees to look for relief from this and future increases. Included were committees on tort reform, on off-shore insurance alternatives, on captive insurance companies, and one to investigate other commercial alternatives.

The first subcommittee found that tort reform was neither an easy nor an effective means to control professional liability costs, at least in the short run. Changes in tort law must apply to all legal areas and not just physician liability. Attempts to change tort law are not well-received by legislators who must think in terms of welfare of injured parties as well as the costs of medical care. Physicians are viewed with suspicion by legislators, many of whom believe that most physicians are wealthy and can well afford high malpractice premiums. Many attorneys have their own self-interested perspective on tort reform for this is a lucrative area in the practice of law.

---

*Connecticut, with a two-year statute of limitations extended by one year for discovery, has an average 5-1/2 year lapse between occurrence and settlement. At the beginning of the premium year the insurance company must guess, with the aid of actuaries, not only the number and nature of the suits that may be filed in the premium year but also for years thereafter. Legal maneuvering may extend the time limitation by years. The insurance company continues to be at risk throughout this "long tail."

Six years after the major crisis of 1975, a considerable experience in tort reform had accumulated. In Indiana a physician-governor influenced legislative enactment of comprehensive tort changes. Many of these reforms fell in court challenges to questions of constitutionality. In spite of the extensive program in Indiana, both the incidence and costs of professional liability suits continued to rise at first, but today the cost for liability insurance in Indiana is well below that in neighboring states. Indiana now has a physician-owned insurance company which started business in mid-1982. In 1979 and 1980 the California legislature also adopted extensive tort reforms. Again, many of the reforms fell in court to questions of constitutionality, and those reforms which were not questioned in court have not had as much effect as hoped on the incidence or cost of professional liability. (The principle constitutional questions have concerned caps on noneconomic awards.) The liability insurance problems are not as extreme now as they were in the 1970s and 1980s. Some court relief has been obtained by legislative support for structured settlements, which reduces the cost of very large suits and in the limitation of noneconomic damages. Although dramatic, high-cost suits occurring infrequently had not contributed greatly to the cost of liability insurance, they are now increasingly common and even little Connecticut has had million-dollar losses in a number of cases in the past five years. Although it was considered essential to continue efforts at tort reform, this was only part of the answer. Other alternatives, such as compensation or no-fault laws, were studied in committees and thought at present to be impractical.

As further increases of 35% in 1980 and 70% in 1981 were proposed by Aetna, negotiations with Aetna moved from the subcommittee and became the primary responsibility of the officers of the Society in cooperation with the Society's staff and the chairman of the Professional Liability Committee. The position of the Society was greatly enhanced by the concurrent decision (see below) to seek a charter for a "captive" insurance company.* The plan finally agreed upon with Aetna for 1981 included a reduction of the premium increase from 70% to 60%, a reduction of agent's commissions from 7.5% to 3.5%, abolition of the contingency fund of 3%, and, most significant, an agreement to return unused premiums and up to 50% of unused investment income.[2] Included also was a three-year guarantee of availability, peer review before cancellation, and increased physician and insurance company involvement in claims management. Aetna employed additional staff in claims management and proposed a program similar to that effectively operated by the New Jersey physician-owned insurance company.

Threatened by the possibility of a CSMS physician-owned company, Aetna insisted that these agreements depend on continued subscription of at least 2,500 physicians. Following the later withdrawal of the Society's endorsement, the profit-sharing feature became inoperative. Whether Aetna's profit-sharing plan would have resulted in retention of a significantly larger part of the market and would have functioned to the advantage of Aetna and the physicians remaining with Aetna is a matter of speculation.

The subcommittee to study physician-owned companies had investigated captive companies, both in-state and "offshore," and possible association with other already formed physician-owned companies such as those in New York, New Jersey, and one from California. The outcome was a recommendation, subsequently approved by the CSMS Council, for a study of the feasibility of forming a captive company in Connecticut. In February 1980 the Professional Liability Committee, now chaired by Dr. Lawrence B. Ahrens, interviewed a number of consultant organizations. It recommended, and the Society approved, the employment of American Health Systems, Inc., of San Francisco, California, (AHS)* represented by John Dorsett.[3] The Society also employed the firm of Milliman and Robertson, Inc., consulting actuaries of Pasadena, California, represented by David Bickerstaff, to do an actuarial analysis to serve as a basis for the feasibility study. To the surprise of the Committee and the Council, the report, given in September, indicated that Aetna's rates, despite the 25%, 35%, and proposed 70% increases, were actually discounted.†[4] Connecticut's undiscounted liability costs could no longer compare so favorably with the rest of the country. AHS recommended that the Society remain for the present with Aetna, considering its favorable premium rates, but should continue to develop a physician-owned insurance company as the most feasible long-term plan.[4] In October 1980 the Council accepted the report and, after considerable discussion, voted unanimously to "Approve the concept, as outlined in Mr. Dorsett's report of 2 October." The Committee on Professional Liability was "Authorized to proceed with American Health Systems to get in place by 1 May alternative plans that could be implemented with dispatch."[5] Meanwhile, negotiations with Aetna were to be continued. In January 1981 the Aetna program, as outlined above, was endorsed by the Society with the understanding by both Aetna and the Society that development by CSMS of the captive company would continue.[6]

---

*A company owned by physician-insureds.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Following the October 1980 action, the Council directed its attorneys to prepare a charter for a physician-owned insurance company, to be known as "Connecticut Medical Insurance Company" (CMIC), for legislative approval. The Society's staff, along with the lobbyist and officers, spent much time developing a plan for presentation to the legislature. At the legislative hearing the major testimony was given by John Dorsett of AHS outlining the CMIC as eventually proposed to the CSMS House of Delegates. Testimony was also given by the Professional Liability Committee chairman, the Legislative Committee chairman, and the president of the Society. Emphasized was the importance of improved physician performance through the efforts of a physician-owned insurance company. No opposition developed and the charter bill was passed by the legislature on the consent calendar.

In the meantime, the Professional Liability Committee was proceeding in other directions. Approached by several insurance agencies interested in administering a captive insurance program, the committee chairman reported to the Council that such agencies would do independent actuarial studies at no cost to the Society. The Council did not object to pursuing this line, and the Professional Liability Committee received a number of reports from a number of actuaries, all but one confirming the Society's figures. The Committee favored the dissenting actuary, who had also served New York and Florida. A brief check, however, revealed that New York and Florida were apparently the only physician-owned companies in trouble in the United States; their difficulties seemed to emanate from under-estimation of premiums from inaccurate actuarial estimates. Subsequent financial difficulties in New York confirmed this impression.

Instead of proceeding with the development of plans with AHS as directed by the Society, the Committee negotiated with several agencies, apparently with a view to recommending a replacement for AHS. The Committee did not meet with AHS or their representatives or engage in any discussions with the AHS, or with Mr. Dorsett, to develop plans for the CMIC.[7] In anticipation of a major role in the development of the captive company for the Professional Liability Committee, the Council appointed an ad hoc committee "to supplement the committee on Professional Liability's negotiations with Mr. Dorsett."[8] As the Professional Liability Committee's actions became increasingly at variance with the Society's intentions, attendance rapidly decreased and the Ad Hoc Committee became inactive. In February 1981 the five members of the Professional Liability Committee still attending meetings, by a three to two vote on a motion made by the chairman, unsuccessfully proposed that the council "withdraw" its unanimous action of the previous October endorsing American Health Systems.[9] Subsequently the Committee recommended to the Society a complex proposal from a brokerage firm using actuarial figures which were inadequate. The premiums as outlined did not provide for adequate reserves and surely would not have been approved by the State Insurance Commissioner. Finally, in late January, in the absence of action by the Professional Liability Committee, the president of the Society, Dr. W. Raymond James, and the chairman of the Council, Dr. Andrew Canzonetti, requested AHS to proceed with development of the CMIC captive according to the plan previously presented to the legislature.

---

*American Health Systems, Inc., was a San Francisco based corporation providing consultative and technical services, primarily (90%) to physicians' organizations, with a principal interest in professional liability.

†Discounted ... the amount of premiums assigned to loss is less than the anticipated loss, the difference to be made up from investment income. Except for the uncertainties of future interest rates and the economy, premiums could be discounted 30-50%. Aetna's rate had apparently been discounted significantly for many years.

In a special meeting the CSMS council approved the AHS' plans, and on 29 April 1981 the CSMS House of Delegates, with no dissenting vote, approved the plans and appointed members to the corporate board and the management committee to organize and manage the CMIC for the Society.[10] At the organizational meeting of the new Council on 20 May, extensive actions were taken to begin the organization of CMIC.

In the summer of 1980, disillusioned by Aetna's agreement with the Hartford Hospital and its medical staff, and aware of physicians' resentment toward Aetna, representatives of the Hartford County Medical Association approached, through the Arthur Watson Agency, other companies including CNA.* The HCMA had had a long and beneficial relationship with the Arthur Watson Company, which served as agent for the popular and successful group insurance plans offered by the Association. In December 1980 CNA representatives contacted state society staff members at the AMA House of Delegates meeting in Chicago. In February 1981 the officers of the state Society met with CNA representatives to review a plan which might be presented to the Society. This proposal offered no advantages over Aetna's. There was no real profit-sharing feature and the proposal offered little in the way of risk prevention and claims programs such as Aetna was proposing. Premium schedules were not then available. At the time CNA had apparently contacted leaders of Health Care, Inc., in New Haven† and offered them premium rates well below the current Aetna schedule. Healthcare did endorse the proposal and CNA then had further conversations with HCMA trustees and made a proposal with very favorable premium rates. The proposal was supported by the HCMA trustees who requested, however, that the plan be offered to the CSMS Council as a statewide plan before final approval. CNA representatives and the Fairfield County Medical Association also began discussion of the proposal. It was then presented in detail to a special meeting of the CSMS Council on 15 June 1981. The plan's premium rates were well below the current Aetna rates. In order to give full consideration to all possible alternatives, the state Society postponed action until the regular meeting 10 days later on 25 June 1981.

Early rumors had suggested that Aetna's premium schedule might be increased by as much as 35% for 1982. Following approval of the CMIC charter by the legislature, it was rumored that the figure would be reduced to something less than 10%. Aetna representatives, however, reported that they would not have rates available before mid-August. In the meantime, AHS was proceeding with the legal and organizational steps necessary to form the CMIC. Unfortunately, the CMIC Management Committee and Corporate Board created by the House of Delegates had not met during the fertile period after the CSMS House of Delegates meeting of 29 April. No preliminary work had, therefore, been undertaken on premiums and rates. Consequently, the Council requested AHS to advise the Council at its 25 June meeting regarding practical premium rates and structures for comparison with the CNA proposal. The day after the 15 June special Council meeting, and nine days before the CSMS Council could act, the HCMA trustees endorsed the CNA plan.

---

*A conglomerate insurance company based in Chicago. The Connecticut Professional Liability Insurance would be placed with the Continental Casualty Company.

†Healthcare, Inc., is an IPA (individual practice association) conducted by physicians of the New Haven County Medical Association.

On 25 June 1981, the AHS representatives reported to the CSMS Council that, in their opinion, the success of CMIC would depend on support of the HCMA and withdrawal of their trustees' endorsement of CNA. The rate schedule recommended by AHS included territorial rating,* a new concept in Connecticut. Since most of the loss statistics in Connecticut were in the hands of Aetna, there was no way of demonstrating the validity of territorial rating, although opinions from Aetna and elsewhere had indicated a much higher loss rate in Fairfield and New Haven counties than in the rest of the state.

Finally, the CSMS Council was faced with four alternatives: first, to continue with the formation of CMIC as directed by the House of Delegates (which would probably depend on HCMA's withdrawing its endorsement of CNA and the approval of territorial rating); second to continue support for the Aetna plan for the coming year, an alternative that was made difficult by the inability of Aetna to commit to a premium schedule at the time; third, to endorse the CNA proposal; and fourth, to postpone any action pending further study and negotiation. Delay would undoubtedly have resulted in continued aggressive marketing of the CNA plan by the Arthur Watson Agency through the county associations. This would have weakened the prospects of success for statewide marketing of CMIC, and probably would have reduced Aetna participation below the 2,500 needed to support the Aetna agreement. Delay also would have reduced the role of the state Society and eliminated the possibility of developing a unified statewide program. To have marketed CMIC under these circumstances would have the state Society competing with the county associations. Not to proceed would leave most of the county associations feeling that HCMA was dictating the state Society's actions.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

After much debate among the councilors centering around the short time interval and the difficulty of organizing CMIC and marketing it against such difficult competition, and the advisability of taking CNAs obviously heavily discounted rates, a motion to request the HCMA trustees to rescind their action was defeated by a close vote. The CNA proposal was then endorsed. Aetna was notified that CSMS endorsement of the agreement with them would be withdrawn as of the end of the year. Development of CMIC was continued[11] and by mid-August the company was organized and placed "on the shelf," ready to begin operation whenever desired.

In August 1981 Aetna began marketing both occurrence and claims-made policies† with territorially-rated premiums and with special classifications favorable to ophthalmologists and several other low-loss groups. Aetna's rates in the small counties were actually reduced and were comparable to the CNA rates. A number of individual physicians, thought by Aetna to be poor risks, were notified that their policies would not be renewed at the end of the year. Now freed from its formal relationship with CSMS, Aetna had apparently opted to compete for low-risk physicians only, and to withdraw from the expensive high-risk areas, a policy which might also have occurred in competition with CMIC.

The Arthur Watson Agency, CNA's managing general agent, proceeded with an aggressive sales campaign headed by Joseph Flynn. By October 1981 some 2,500 subscribers had transferred to CNA. This number was sufficient to support the endorsed agreement that rate increases would not exceed 35% for the following policy year.

For the year 1982-83 the IPA members in New Haven County continued to have more favorable CNA rates than other physicians in the county or in the state. The HCMA CNA plan was the same as the statewide plan. The Hartford Hospital staff continued with the special Aetna plan. The revised CNA rate schedule for 1983 was increased an average of 24% with significant further changes in some categories. There was still no territorial rating and the

---

*Territorial rating is a method of pricing insurance by dividing each state into geographic regions and setting rates for each region to reflect previous regional loss experience.

†"Occurrence" policies insure for events that occur during the policy year regardless of when the claims are made. "Claims-made" policies insure for claims made during the policy year regardless of when the event occurred. "Modified claims-made" is a claims-made policy which insures future claims in case of death or disability and, in defined cases, retirement.

CNA commitment was to continue one more year before negotiating such changes. While the rates in some categories and in the four smaller counties were competitive, the 1983 Aetna territorial rates generally remained well above CNA's. Other insurance companies appeared in the state, notably St. Paul Insurance Company, which began aggressive marketing of claims-made policies to groups and individual physicians. Obstetricians and psychiatrists had national plans favorable to their specialties.

The 10-year involvement of the Connecticut State Medical Society in a unified professional liability program was a positive experience in spite of expenditures of more than 300,000 dollars. The availability of insurance for members, and perhaps also the enhancement of the visibility of the state Society, resulted in a 25% increase in membership with the largest increase during the 1975-76 crisis. Premium rates during this decade were low compared to the rest of the country. The increased competition, occasioned by the chartering of the CMIC, and negotiation with the various insurance companies, resulted in premium reductions which saved many millions of dollars for Society members. The Society made progress in areas of physician participation in claims management and in risk management. A large pool of knowledge concerning professional liability was acquired by the staff and by the leaders of the Society.

The year 1982 saw a moratorium in Society activities in professional liability. There would be some time before experience with CNA's claims-management program accumulated. There was little CNA development of risk-management programs. The CSMS claims review panels continued to function for Aetna claims. County level claims review and insurability review committees for CNA were formed and were active but with varying impact on CNA actions. The Joint Commission on Accreditation of Hospitals (JCAH) continued to push for risk management through quality assurance committees in hospitals, a program not then notably active or effective in Connecticut.

In 1982, however, Connecticut appeared to be in a weaker position than in 1971. There was a statewide endorsed plan with rates that were favorable, or at least discounted significantly, but little in that plan that offered any hope that the steadily increasing incidence and cost of professional liability suits would not continue. Reasonable estimates are that only 5% of potentially compensable events result in claims. The potential for increase in suits was enormous. Neither was there reasonable expectation that costs of claims would not continue to escalate.

It appeared unlikely that legislative changes would make professional liability a manageable problem. The only alternatives open to physicians were claims and risk-management programs that would convince the public that physicians were competent and were policing their own members, thereby reducing the number of potentially compensable events. It is in these areas that captive companies offer the most promise since the company itself has both the framework and organized motivation to promote effective risk-management programs. It was the national experience of those years that most risk-management programs and most improvements in claims management came about through the efforts of physician-owned insurance companies. By the early 1980s over half of all physicians in the Unites States were insured through physician-owned companies. With the possible exception of New York State, these companies have been stable and successful.

Our experience during those years emphasized the difficulty of organizing a captive insurance company. It was surprisingly easy for CNA to divide the state by splitting off the New Haven IPA and the HCMA. It was disappointing to have the Professional Liability Committee working at cross purposes with the state Society and significantly impeding the development of the CMIC.

Experience in other states had led the Society to expect strong competition in starting a captive company, most of this competition was expected to come from Aetna. In the 20 May 1981 Council meeting a strong feeling of commitment to the development of CMIC regardless of competition was evident. It was expected that there would be rate cutting by the competition. The Council was committed to the idea that a company must be soundly financed, solidly based, and well capitalized and, in the beginning, higher premiums might be required. Within a few weeks, this commitment wavered in favor of short-term expediency. It seemed wiser to let CNA take the loss in their first year, as they surely did, than for a new captive company to start with a losing year.

With the high interest rates of the 1980s the large cash flow of liability insurance was attractive to insurance companies and the Society was believed to be able to maintain competition which would continue to bring premiums comparable to those of the captive companies. Although there were sporadic stirrings of interest in CMIC, no definite plans were forthcoming to activate the company during the next three years.

It was inevitable that CNA would raise premiums enough to prevent further losses after what must have been a disastrous first year for them. The Society was in a good position with CMIC created and ready to go and aware that CMIC could succeed with premiums at least 10% lower than the commercial companies. It was only a matter of a few years before all the necessary conditions for success would be fulfilled.

REFERENCES

1. Aetna, "Report to the Society," August 1980.
2. CSMS HOD HANDBOOK, Wednesday, 29 April 1981, "Report of the Committee on Professional Liability."
3. Minutes of the Council, CSMS, 15 February 1980.
4. Report to CSMS by American Health Systems, 16 September 1980.
5. Minutes of the Council, CSMS, 15 October 1980.
6. CSMS HOD HANDBOOK, Wednesday 29 April 1981, "Report of the Committee on Professional Liability."
7. Minutes of the Committee on Professional Liability from 16 September 1980 through 14 April 1981.
8. Minutes of the Council, CSMS, 15 October 1980.
9. Minutes of the Professional Liability Committee, 18 February 1981, and Minutes of the Council, CSMS, 18 February 1981.
10. Proceedings of the House of Delegates; CSMS; 29 April 1981.
11. Minutes of the Council, CSMS; 25 June 1981.

HEALTH SCIENCES LIBRARY UNIVERSITY OF MARYLAND BALTIMORE

Guest Editorials

Preventive Medicine for Senior Citizens: Uncovered

The routine annual physical examination has been a time honored component of medical practice, although recently its benefits have been called into question. The American College of Physicians, United States Preventive Services Task Force, and others have suggested guidelines for performing selective preventive medical examinations and counseling depending on the patient's age and risk factors.[1] However, the health insurance carrier for senior citizens, Medicare, does not mandate payment for preventive service. Services that Medicare never pays for are called "noncovered" (such as influenza vaccinations or "routine examinations"). The controversy may begin when a physician performs a comprehensive examination of a patient with multiple chronic medical problems that require monitoring of the disease status and any reactions to treatment, especially when that patient is asymptomatic at the time of the visit. Although such an examination is warranted and constitutes the practice of quality medical care, is this a covered Medicare service?

According to the CPT guidelines for coding evaluation and management services for office visits for established patients, two of three components are required to establish a level of coding; history, examination, and medical decision making.[2] The "nature of the presenting problem" is considered a contributory factor, not a mandatory one. However, Medicare may interpret this differently. Medicare may consider the "nature of the presenting problem" as paramount, in addition to the "key components." For example, imagine a patient with diabetes mellitus, hypertension, stable angina following angioplasty, and osteoarthritis with occasional episodes of gout who is controlled with five different medications. He is scheduled for a comprehensive examination but he happens to feel well at that time. The physician performs a comprehensive history and examination and reviews data that may be of moderate complexity (CPT code of 99215 , two of three key components met establishing the highest level of service). If Medicare were to review the chart they may consider this "routine" and deny payment for the visit at that level of coding because the patient did not have an active symptom to justify that intensity of service. Of course if the patient was in acute congestive heart failure, diabetic ketoacidosis, or experiencing crushing chest pain it would be difficult to get a complete social history and review of systems to satisfy the CPT coding requirements.

The recently published issue of *Medicare News*, briefly speaks to this topic as "Issue 5," but may generate more questions than answers.[3]

> Billing for annual routine (asymptomatic) examination in conjunction with a medically necessary visit.
>
> DECISION:
>
> 1. The annual asymptomatic examination is a noncovered service and the physician may bill the patient, who is responsible for the payment.
>
> 2. If there is a symptom or complaint or medical condition being followed in the course of the examination, then the appropriate level of visit code should be used to report the covered encounter and the limiting charge rules apply. The visit or consultation cannot be carved into covered and noncovered portions.
>
> 3. There could be covered and noncovered procedures performed during a covered E/M service (eg, screening x-ray, EKG, lab tests, etc.). These would be considered individually.
>
> a. Those which are for screening for asymptomatic conditions would be considered noncovered.
>
> b. Those ordered to diagnose or monitor a symptom, medical condition, or treatment would be evaluated for medical necessity.
>
> 4. The physician would be responsible to notify the patient in advance of his or her liability for the charges for nonmedically necessary services.[3]

What is the proper coding and billing procedure for Medicare patients for a comprehensive visit who have no active symptoms or diseases identified to justify that level of coding? One approach may be to inform the patient at the time the visit is scheduled that this is a service not covered by her insurance, although it may be medically recommended. Inform the patient that she will be responsible for the standard office charges for the visit and all ancillary tests (under these circumstances one is not restricted to Medicare fees and may charge the regular office fee, but it may be advisable to accept the Medicare allowed amounts for laboratory tests and ancillary ser-

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

Regarding Payment for Complete Physical Examinations with Medicare Insurance

Dear Patient,

A comprehensive physical examination is an important aspect of medical care that screens for unseen health conditions that may be successfully treated when found early. Although the advantages of preventive health care are well known, by law, Medicare does not pay for this important aspect of medical care. Another example of preventive medical practice that Medicare does not cover is the annual flu shot, universally recommended to everyone over age 65. Although Medicare acknowledges that it may be good medical practice to offer these services, current law and new codes required this year, do not provide for its payment.

We continue to encourage our Medicare patients to have annual comprehensive examinations, however Medicare will not pay for these visits until the law is changed. Therefore we will continue to perform these services but we must bill you directly and the fees will NOT be reimbursed by Medicare.

The fees for a comprehensive examination are as follows:

Complete review of history and physical examination:	$—.—
(Preventive examination codes 99397 or 99387)	
Electrocardiogram:	$—.—
Urine test:	$—.—
Stool cancer screening test:	$—.—
Complete blood count (anemia test):	$—.—
Blood drawing and preparation fee:	$—.—

We will submit the charges to Medicare for these services. The preventive care visit is not covered by Medicare and you must make payment at the time of your visit. If Medicare pays for the laboratory charges or EKG we will accept that payment. If Medicare does not allow those services then you will be billed for them at the Medicare allowable rate.

You must carefully read the following statement, sign and date it, and mail it back to us in order for us to schedule you for your complete physical examination:

"Medicare will only pay for services that it determines to be 'reasonable and necessary' under section 1862(a)(1) of the Medicare law. If Medicare determines that a particular service, although it would otherwise be covered, is not reasonable and necessary under Medicare program standards, Medicare will deny payment for that service. I believe that, in your case, Medicare is likely to deny payment for the comprehensive examination and related services because 'routine' services for preventive care are not covered .

Beneficiary agreement: I have been notified by my physician that he believes that, in my case, Medicare is likely to deny payment for the services identified above for the reasons stated. Being that Medicare would deny payment, I agree to be personally and fully responsible for payment."

Patient's Signature ____________________ Date ____________

vices). Although it is not required to obtain written notification from the patient for a "noncovered service" (ie, Preventive Medicine service codes 99381 through 99397 and associated tests) it may be prudent to do so. Above is a sample disclosure that may provide a useful guide. The language is adapted from Medicare regulations.

The more difficult issue arises when the patient has many active but relatively asymptomatic medical problems and comes for a comprehensive visit. If one bills for a level five visit (ie, 99205 or 99215) it may be wise to have the patient sign a notice informing him that Medicare may deny payment for that service for the stated

Medicare Notice

"Medicare will only pay for services that it determines to be 'reasonable and necessary' under section 1862(a)(1) of the Medicare law. If Medicare determines that a particular service, although it would otherwise be covered, is not reasonble and nessary under Medicare program standards, Medicare will deny payment for that service. I believe that, in your case, Mecicare is likely to deny payment for the following services(s):

Medicare may deny payment for the above service because:

[ ] routine services for preventive care are not covered.

[ ] services at this frequency may not be covered.

[ ] services may have been provided in a facility providing a less acute level of care.

[ ] ____

Beneficiary agreement:

I have been notified by my physician that he believes that, in my case, Medicare is likely to deny payment for the services identified above for the reasons stated. If Medicare denies payment, I agree to be personally and fully responsible for payment."

____ ____________

Patient's Signature Date

reasons but the physician, nevertheless, will bill Medicare for the visit. This is a covered service, under appropriate circumstances, but may be viewed by Medicare reviewers as "not medically necessary." If upon chart review Medicare denies payment for that level of coding then the physician may bill the patient for the difference.

The "Medicare Notice" (above) may be adopted for that approach. Alternatively the physician could notify the patient before the visit is scheduled that this is not a covered Medicare service and bill it as a Preventive Medicine service to the patient directly, as described in the previous paragraph. The topic of the comprehensive annual examination remains a controversial one for patients with Medicare insurance given the current interpretation by HCFA of the CPT Evaluation and Management codes. Physicians should review the AMA CPT 1993 guide and Medicare literature and develop an office policy that properly informs patients of their liability, fairly compensates the physician, avoids the risk of a Medicare denial and, most importantly, offers quality medical care.

Paul Dolinsky, M.D.

West Hartford

REFERENCES

1. Hayward RS. Stenberg EP: Preventive Care Guidelines: 1991, *Ann Int Med* 1991;114:758-88.
2. American Medical Association, CPT 1993.
3. The Travelers: *Medicare News*, 12/92.007:10.

Guest Editorials continued next page

Age-Related Macular Changes: A Plea

We should drop the terms senile macular degeneration, age-related macular degeneration, and age-related macular disease, substituting age-related macular changes, or retinopathy. Better yet, just changes.

“Senile” is demeaning. “Degeneration” is frightening and almost akin to “cancerous.”[1]

Nor should one speak of age-related macular “disease.” It is not infectious or “catching.” The macular is the central part of the retina. The very center is called the fovea. Usually, the peripheral part of the retina and peripheral vision are spared. It may be hereditary, due to trauma, or to other conditions as yet not clearly understood. It may be referred to by ophthalmologists as “dry” or “wet,” depending on whether there is fluid near the macular. The latter is relatively rare and is amenable to laser treatment if it does not spontaneously absorb.

“Senile”? It can occur in the 40s, 50s, or 60s, at which time people are otherwise physically and mentally healthy, just as they may be in the 70s, 80s, or 90s.

Treatment: Laser treatment of a leaking vessel has been mentioned. Extrafoveal neovascularizatin responds to treatment. Interferon and antiangiogenic substances, and submacular surgery offer hope.[2,3,4]

Prevention: Avoiding prolonged and unprotected exposure to bright sunlight is wise.

In 1963, Margaret J. Albrink, then on the Yale medical service, and I, as chief of the eye service, reported in the *American Journal of Ophthalmology* our studies on serum lipids as the possible culprit.[5] However, we found no correlation and at that time advised patients that if they smoked, they should stop. We also added that they take one multivitamin at meal time with the hope that it might bring extra nutrition to a relatively avascular area. Today, many ophthalmogists recommend a multivitamin with just the “correct” addition of zinc and trace metals. Although “the jury is still out,” I myself practice what I preached years ago. The cost is minimal and if for no other reason, and until we can find something better, it does offer hope.

The past and the future: Will it be possible to transplant a whole eye or retinal cells to the macular?

Years ago I had learned languages with the hope of becoming a diplomate. Instead I became a doctor, but my knowledge of languages was put to good use. Professor Leon Stone, my teacher at Yale, taught me a very important lesson. “Never do anything without returning to the original source.” If in doubt, he would repeat the whole experiment, and my translations often helped. Under the microscope he transplanted whole salamander eyes (*Triturus viridescens*) in 177 operations. Ninety-four eyes showed return of vision. This work, “the discovery of the decade” won him the coveted Doyne medal at Oxford, England.[6] This work has helped in the understanding of repair and healing of severed nerves . Although transplantation of corneas in human beings has been successful, I do not foresee transplantation of the whole human eye in our life-time. Yet, there has been work in this area in transplantation of cells to the retina.

“We are constantly trying to treat macular changes and their sequelae…” wrote a pioneer in this area, Gholam A. Peyman, at LSU on 10 February 1993. “Unfortunately, we are not there as yet.” I made the bold suggestion that he transplant healthy young retinal cells early, and add “growth factor”[7] which is presently very expensive. Early, before scar tissue has formed and before connections to the visual cortex and the cells in the cortex cease to function. I made the crude analogy of the futility of watering a tree when the roots had died. “That’s then too late,” I added.

Conclusions: Age-related macular change (AMC) is the best way to describe changes in the macular area usually found in older individuals.

“Don’t,” said my friend Bernie S. Siegel, M.D., quoting Norman Cousins, commit “psychological malpractise.… Don’t take away hope… tell the truth with hope.”

R.M. Fasanella, M.D.
Eye Research, Section of Ophthalmology
Hospital of St. Raphael, New Haven.

REFERENCES

1. Jamposky A: Senile accomodative degeneration (SAD). *Am J Ophthalmol* 1991; 112:510.
2. Fung WE: Interferon alpha 2a for treatment of age-related macular degeneration. *Am J Ophthalmol* 1991; 112:349-50.
3. Guyer DR, Adamis AP, Gragoudas ES, et al: Systemic antiangiogenic therapy for choroidal neovasculariation: What is the role of interferon alfa? *Arch Ophthalmol* 1992; 110:1383-4.
4. Guyer DR, Tiedeman J, Yannuzzi LA, et al: Interferon-associated retinopathy. *Arch Ophthalmol* 1993; 111:350-6.
5. Fasanella RM, Albrink MJ: Serum lipids in patients with senile macular degeneration. *Am J Ophthalmol* 1963; 44:709-13.
6. Stone LS: Return of vision and functional polarization in the retinae of transplanted eyes. *Trans Ophthal Soc UK* 1947; 67:349-68.
7. Glaser BM, Michels RG: Induction of a “retinal patch” by transforming growth factor-beta in the treatment of full-thickness macular holes. *Invest Ophthalmol Vis Sci* 1991; 32 (Suppl):713.

Guest Editorials continued next page

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

OREF

The 1992 Campaign of the Orthopedic Research and Education Foundation (OREF) in Connecticut has come to a close with new levels of giving having been established. This year, forty-two Connecticut orthopedic surgeons and one friend of orthopedics have contributed at the Order of Merit level (gift of $1,000 or more) to rank 17th among the fifty states.

Nationally, the campaign has raised more than three million dollars. Fifty-eight new research grants for 1993 have been approved and thirty-eight grants have been continued. In addition, the OREF will again support the Residents Conference oft he American Orthopedic Association and will for the first time fund a major Fellowship in Health Services developed through the American Academy of Orthopedic Surgeons.

Below is a list of this year's contributors. An asterisk indicates those who participated as local campaign chairmen.

Dr. & Mrs. Alfredo L. Axtmayer, Wallingford*
Dr. & Mrs. Peter R. Barnett, Hartford
Drs. Gerald J. Becker & Sheryn D. Holm, Glastonbury
Alan E. Bernstein, M.D., Middletown
Dr. & Mrs. C. Robert Biondino, Meriden
Bruce D. Browner, M.D., Farmington
Patrick J. Carolan, M.D., Bridgeport
Dr. & Mrs. Chang Song Choi, Bristol
Eui Kyun Chung, M.D., Putnam*
Edward J. Collins, Jr., M.D., Willimantic
Dr. & Mrs. Thomas W. Dugdale III, Hartford
Dr. & Mrs. Malcolm S. Edgar, Jr., Norwich*
Dr. & Mrs. Robert L. Fisher, Hartford
Gary E. Friedlaender, M.D., New Haven
Dr. & Mrs. Richard L. Froeb, New Britain*
John P. Fulkerson, M.D., Farmington
Harry R. Gossling, M.D., Farmington
Dr. & Mrs. Robert A. Green, Hartford
Mr. Charles A. Heimbold, Jr., Riverside
Peter Jokl, M.D., New Haven
David B. Kalayjian, M.D., Middletown*
Kristaps J. Keggi, M.D., Waterbury*
Dr. & Mrs. W. Jay Krompinger, Hartford
Gerard J. Lawrence, M.D., Willimantic*
Courtland G. Lewis, M.D., Farmington
Dr. & Mrs. Richard M. Linburg, Hartford
James K. Lynch, M.D., New Haven
Dr. & Mrs. Frank W. Maletz, New London*
Dr. & Mrs. John J. O'Brien, Hartford*
Herbert S. Pasternak, M.D., Hartford
Richard R. Pelker, M.D., New Haven
Dr. & Mrs. John F. Raycroft, Jr., Hartford*
Thomas S. Renshaw, M.D., New Haven
Dr. & Mrs. William G. Richeimer, Norwich
Pedro A. Romero, M.D., Manchester
Dr. & Mrs. Vincent M. Santoro, Hartford
Dr. & Mrs. Francis P. Saunders, Danbury*
Dr. & Mrs. Steven F. Schutzer, Hartford
Dr. & Mrs. Enzo J. Sella, Hamden*
Dr. & Mrs. Kevin P. Shea, Farmington
Wayne O. Southwick, M.D., Old Lyme*
Robert A. Stanton, M.D., Fairfield
Dr. & Mrs. Gordon A. Zimmermann, Hartford

These leaders and the other contibutors in the state should be congratulated for their continuing generosity, particularly in these uncertain times.

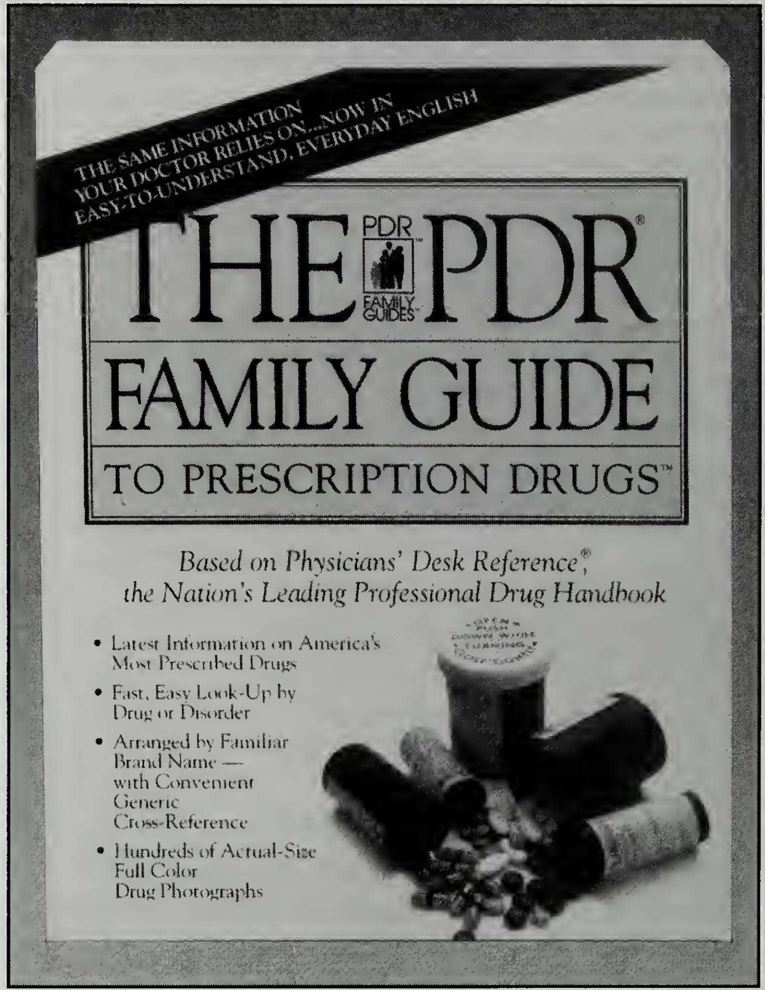
John F. Raycroft, M.D.
Hartford

Physician's Desk Reference®
Releases New Consumer Guide
Helps People Become Medically Literate

Health-conscious Americans are demanding up-to-date information on how the medications prescribed for them impact their bodies. They want a dialogue with their doctors; they insist on participating in the decisions that affect their health. Many have purchased for their home bookshelves the same reference book that physicians have relied on since 1947, Physician's Desk Reference® The PDR is the only drug reference available that gives complete, FDA-approved guidelines for safe and effective use of prescription drugs.

The **PDR® Family Guide to Prescription Drugs™** (ME Data, $24.95) draws on the same authoritative facts found in the PDR. It is the only drug handbook that lists drugs under their familiar brand names (ie, the way consumers commonly refer to them), then cross-reference every brand by generic name and indexes each drug by the diseases for which it is prescribed. This makes the Guide simple to use and permits fast, easy look-ups. In addition, it includes overviews of our most common diseases and disorders, and the treatments we have available today.

Designed to enhance the doctor/patient relationship, the **PDR Family Guide to Prescription Drugs** reminds us of the questions we need to ask our doctors and pharmacists, and the basic instructions and cautions we must remember when taking prescription medications. It helps us use medications safely and effectively, alerting us to the conditions that increase the chances of a possible adverse reaction. And perhaps most important, it highlights the medical problems and conditions that might interfere with a prescription—facts about our personal health that we must make certain the doctor has in order to assure the safest, most effective treatment.


THE SAME INFORMATION YOUR DOCTOR RELIES ON...NOW IN EASY-TO-UNDERSTAND, EVERYDAY ENGLISH
THE PDR® FAMILY GUIDE TO PRESCRIPTION DRUGS™
PDR FAMILY GUIDES
Based on Physicians' Desk Reference®, the Nation's Leading Professional Drug Handbook
• Latest Information on America's Most Prescribed Drugs
• Fast, Easy Look-Up by Drug or Disorder
• Arranged by Familiar Brand Name — with Convenient Generic Cross-Reference
• Hundreds of Actual-Size Full Color Drug Photographs

PDR® FAMILY GUIDE TO PRESCRIPTION DRUGS™
ME Data — $24.95 (softcover)

In addition to its easily-accessible, dictionary-style arrangement of drug profiles and its unique disease and disorder index, the **PDR Family Guide** includes hundreds of full-color, actual size photos of the leading brand name and generic prescription products. These can be a lifesaving aid for consumers who have mixed up their medications.

Appendices include a directory of regional poison control centers and hotline phone numbers, as well as lists of drugs that meet certain conditions, such as those that are sugar- or alcohol-free and those that may make you unusually sensitive to light.

Today, more than ever before, we are aware that we as individuals have the responsibility and the right to participate in our own health care. Modern drug therapy is a vast and complicated field—so complicated that, for many questions about medicines, the answer varies from patient to patient. The PDR Family Guide to Prescription Drugs is an aid in the cooperative effort with our doctors, alerting us to the most pertinent questions to ask and clarifying the answers we receive, thereby providing us with the tools we need to supervise our won medical care as effectively as possible.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

50 Years Ago

From *The Connecticut State Medical Journal*

May 1943

Connecticut Colonial Medicine

HOWARD W. HAGGARD, M.D., New Haven

The Author. *Professor of Applied Physiology, Yale University*

Presented at the Sesquicentennial Celebration of the Connecticut State Medical Society, Middletown, June 4, 1942.

THE story of colonial medicine in Connecticut has often been told and it has been told especially well by men who are in the audience today. Usually, and naturally, the emphasis has been upon the lives of those who have practiced the healing arts. This morning, in contrast, I shall omit the biographical and emphasize instead the cultural background and of it, especially those influences which gave distinctive characteristics to Connecticut colonial medicine.

But let me, first, in a few words set up some land marks and some time marks. And forgive me if, in doing so, I retell what you knew in greater detail as grade school students.

As you are aware, our state owes its settlement, in part at least, to the fact that certain of those who came in the Puritan migration of 1630 were unhappy in our older sister colony of Massachusetts. In 1633, members of the Plymouth Colony established a trading post near Windsor and John Oldham explored the valley and reported upon its resources. So favorable was his report that, as you will recall, the inhabitants of three Massachusetts towns, Watertown, Dorchester, and what is now Cambridge, migrated here. Those from Watertown founded Wethersfield in the winter of '34-'35; those from Dorchester settled in Hartford the autumn of '35 and those from Cambridge in Windsor in the summer of that year. They moved to Connecticut because of their discontent with the autocratic character of the government of the Massachusetts Colony. But once removed, the instrument of government they adopted here differed in few particulars from that of Massachusetts. Still another contingent settled near the mouth of the Connecticut River in '35 under the auspices of an English company whose leading members were Lord Say and Sele and Lord Brooke—hence the fort and town of Saybrook.

The primary migrations of 1634 and 1635 were followed by others in rapid succession. There was the New Haven Colony in '38 which soon spread to include Milford, Guilford, Stamford and Southold on Long Island and then Branford. The next important step toward the formation of modern Connecticut was the union of New Haven and the older colony under the charter of 1662.

My time marks thus begin with the 30's of the 17th century and they should run into the 70's of the 18th century. But I shall not go that far; my intention is rather to try and define something of the early forces which gave shape to Connecticut medicine.

Now as to my land marks: The boundaries of Connecticut were once defined as extending west from Narragansett Bay to the Pacific Ocean. But I shall limit myself, as did the colonists, more nearly to the boundaries of the state as we know them now.

The physical geography of our countryside is different from that of Massachusetts; and it is different from that of New York. And the nature of a land appears to make a lasting impression on the inhabitants. Or it may be possible, instead, that certain characteristics of men guide their choice of the land in which they wish to live. Whichever it is, the colonists of Connecticut and their doctors had characteristics different from those of the people and doctors of Massachusetts and of New York. It is probable that the first application of the word "Yankee"

From *The Connecticut State Medical Journal*, May 1943.

was to the colonists of Connecticut and that from their characteristics, in turn, it took on some of its subtler meanings. One of the best expressions of the distinctive characteristics of the colonists of Connecticut—and of their doctors—is to be found in the none too idealistic statement in which they themselves defined their own code of behavior. I quote: "... to avoid notoriety and public attitude; to secure privileges without attracting needless notice; to act as intensely and vigorously as possible when action seemed necessary and promising; but to say as little as possible, and evade as much as possible when open resistance was evident folly."

You can see those same characteristics, clear and unmistakable today, in the older inhabitants at a town meeting in any rural community of Connecticut—and in any successful horse trader.

With these shrewd characteristics there was another; it was one rarely possessed by the doctors of Europe from whom those of the colonies stemmed. It was a religious, a spiritual quality, which was probably, in part, an outgrowth of the fact that often the colonial doctor was the preacher and the preacher the doctor; and, in part, it may have been an expression of the deep insecurity that all men must have felt in the new land. Dr. Thoms, in his essay on medicine in the New Haven Colony, has expressed admirably this quality in speaking of the favorite remedies of colonial physicians and specifically of Winthrop's Rubila. He says, "While the doctor of that day had undoubted faith in these unusual remedies, we must not forget that he had greater faith in God, not only as the ruler of the Universe, but as the controller and participant in every act in daily life. The Puritan mind trained to see the spiritual in everything saw in disease, not the breaking of Nature's law but the manifestation of a wrathful Divine Hand."

In contrast the contemporary physician living in the cities of Europe was more prone, and perhaps more correct, in attributing many diseases, at least the non-epidemic ones, to the dissipations of his patients and to offer tolerant and sophisticated advice along with his secret pill. The colonial physician saw sin and prescribed repentence with his simples.

It is easy, as we gather here, to look back with some condescension at the doctor of early colonial times. He and his patients are to us impersonal; because of the gap of time and of material and intellectual civilization which separates us, we cannot identify ourselves with him or them. But those doctors were men who worked as earnestly as any one of you today to save life, to prolong life, and to ease suffering. And their patients were not impersonal if forgotten characters of history; they were men and women and children who loved and were beloved and who had the same fears and suffering that your patients have today.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Place yourself in the position of the doctor of colonial Connecticut. You will find that you are not practicing a specialty, but practicing, in combination, medicine, surgery, pharmacy and dentistry—and that in a day when surgery and dentistry were beneath the dignity of the physicians of Europe. And you will find that you are practicing all four unremuneratively so that to support your family you must have still other callings—that of merchant, mechanic, artizan, school teacher or preacher. There is no hospital, few of the medicaments you use today are available, and what medicaments you do use you must usually prepare yourself from the plants and barks and berries of the countryside. You make your rounds, not through wards in an institution, not by travelling in an automobile, but on horseback over rough trails and across unbridged streams in a sparsely settled and wild countryside.

Picture yourself in such surroundings with none of the diagnostic instruments you now possess and with only the tiniest part of the knowledge you now have and among patients who suffer from every illness, every injury and every misfortune which you know today and many that you rarely see. My sympathy goes to you, gentlemen, in such surroundings—but it goes even more to your patients as you do your surgery in the cabin with no knowledge of pathology, of antisepsis and asepsis, and of anesthesia; as you deal with malaria and diphtheria without specifics and with no knowledge of the principles of infection; as you cope with consumption even before the pathology of the disease has been defined; as you try your remedies in the control of widely prevalent smallpox, typhoid and dysentery and even rabies with no knowledge of sanitation and prophylaxis. Men who could practice under those conditions and retain the love and respect of their patients, and who could bring actual benefit to their patients, were indeed great men of medicine.

It was with intent that a moment ago I emphasized the peculiar combination of spiritual and practical qualities of the early doctor of Connecticut. In him, as an individual, there was almost typified the two great streams of medical philosophy which, with the coming of the settlers, actually met in their extremes of practice in the lands of Connecticut. One of these streams was the lore of the Indian; the other was the learning of the European. The refined spiritual beliefs of the settler tinctured his medicine even as some empirical knowledge tinctured the crude spirit medicine of the savage. The lore of the Indian was the ancient spiritualistic medicine which was the inevitable development of all untutored people. Under its philosophy, disease was the result of the machinations of malign supernatural creatures and of magic. The essence of the medicinal treatment was psychotherapy. But it extended to include a crude surgery which was too often turned to mutilation rather than repair, an especially robust physiotherapy, a hygiene of taboos, an education in the priestly medicine and a drug therapy which included the herbs—even to hemlock buds for scurvy—and animal flesh and oil. It is this drug therapy which has survived in our memory as the famous Indian remedies sold in our boyhood by the peddler and the street corner faker and it is a therapy different mainly in its principles from that of Galen.

The learning of European medicine which the colonists brought with them was a branch from that main stream of the enormously ancient spiritualistic medicine possessed by the Indian. The medicine of the Indian was no different in principle from that of the ancient but civilized Egyptians and of the Greeks up to some 2,000 years before the days of our colonies.

At that time the new stream branched from the old. It started with the separation of medicine from religion at the hands of the Grecian philosophers and the equally important separation of the philosophers from medicine by the physicians whom we revere under the name of Hippocrates. It was a medicine in which man sought to work out his salvation on earth by his own observations and reasonings. It is the stream of medicine which we follow today; it is now wide and deep but mainly its width and depth have come, not from new philosophies but from well springs of factual knowledge arisen long since the period with which I deal. In the time of the Connecticut colonists the philosophy of medicine was different from that of the savage but any advantage of this difference to the patient was perhaps greater in principle than in fact. The factual medical knowledge was pitifully meager. It carried with it much of the flotsom of the Middle Ages.

You will recall that it was less than a century before the colonists settled in Connecticut that Vesalius founded modern anatomy; that it was in the same period that Paré raised surgery from a trick of barbers, executioners and mountebanks to at least a manual art from which, for want of interest, it then declined. Forty years before the colonists, Harington, the godson of Queen Eizabeth, invented the water closet—which certainly no colonist of Connecticut ever saw. Seventeen years before the colonists, the first edition of the London Pharmacopoeia was printed. And only seven years before the colonists, Harvey published his discovery of the circulation of the blood.

The colonial physician had something of the philosophy of Hippocrates. He had the gentle herb therapy of Galen cautiously accentuated here and there with the harsher mineral medicaments of Paracelsus. He had some fairly sound knowledge of anatomy from the heritage of Vesalius; he had a little of the common sense in surgery from Paré and his followers; and he had a smattering, no more, of the sciences that were generating in the universities of Europe.

It was not so much in useful knowledge as in his attitude and behavior that the colonial doctor differed from most physicians of Europe. Across the ocean, too often the little knowledge was overcompensated by a show of charlatanism which, in coming years, Molière was to ridicule in France. Fortunately, unlike the physicians whom Molière goaded with his barbed pen, the doctors of Connecticut had no appreciative audiences for medical theorization. Life was hard; and it was serious. The doctor here had no opportunity to over-compensate for his lack of knowledge by the extravagant attire and arrogant manners of many of the English practitioners of his time. Connecticut of that day was hardly the place for such ostentation.

Far more often than not the colonial physician did not attend a medical school—there was none in this country until well into the 18th century. Some young men went back to Europe for their medical education but most learned their healing by apprenticeship and got their grant to practice on application to the General Court. The apprentice lived at the doctor's house, he cared for his horse and rolled his pills. He read what few books were in the doctor's library. But his real training was gained when he rode with the doctor on visits to his patients. There at the bedside the older man pointed out the symptoms of disease and indicated the treatment. As they rode from one home to the next, they discussed the case and there was plenty of time for both discussion and thought. It was clinical instruction. And it was instruction by doctors who had those characteristics common to the people of Connecticut. From it, to paraphrase a quotation I used earlier, there came a medicine without notoriety and public attitude; a medicine intended to secure benefit without attracting needless attention; and a medicine applied as intensely and as vigorously as possible when action seemed necessary and promising.

A moment ago I drew a contrast between the medicine of the Indian and the medicine of the Colonial physician. My words were general—let me now make them specific with two examples, both from writers of the period with which I deal.

The first is Lawson's description of an Indian medicine man in therapeutic action. He says: "As soon as the doctor comes into the cabin, the sick person is set on a mat or skin stark naked except for some trifle. In this manner the patient lies when the conjurer appears and the king of that nation comes to attend him with a rattle made of a gourd with peas in it. This the king delivers into the doctor's hand whilst another brings a bowl of water and sets it down. Then the doctor begins and utters some words very soft; afterward he smells the patient's naval and belly; andsometimes scarifies him a little with a flint, or an instrument made of rattlesnake teeth for this purpose; then he sucks the patient and gets out a mouthful of blood and serum—which be spits in the bowl of water. Then he begins to mutter and talk apace, and at last cuts capers and claps his hands on his breeches and sides till he gets into a sweat, so that a stranger would think that he was running mad, now and then sucking the patient— still continuing his grimaces and antic postures.... At last you will see the doctor all over of a dripping sweat and scarcely able to utter one word, having quite spent himself; and then he will cease for awhile, and so begin again till he comes in the same pitch of raving and seeming madness as before; all this time the sick body never so much as moves, although doubtless the lancing and sucking must be a great punishment to them.... At last the conjurer makes an end, and tells the patient's friends whether the patient will live or die; and then one that waits at this ceremony takes the blood away— and buries it in the ground in a place unknown to anyone but he that inters it."

Now, as a contrast, let me recount in a few words, and again in quotation, this time from Governor Winslow in the "Good News from New England," the treatment of Massasoit by Winslow himself. He says:

"When we came thither we found the house so full of men as we could scarcely get in; though they used their best diligence to make way for us. They were in the midst of their charms for him, making such a hellish noise, as to distemper us that were well."

Winslow finally made his way through the milling Indians to the chief's side and offered help. The chief nodded. "And," says Winslow, "having a confection of many comfortable conserves, etc.; on the point of my knife I gave him some; which I could scarcely get through his teeth. When it was dissolved in his mouth, he swallowed the juice of it whereat those that were about him much rejoiced saying, he had not swallowed anything in two days before. I then desired to see his mouth which was exceedingly furred; and his tongue swelled in such a manner, as it was not possible for him to eat such meat as they had, his throat being stopped up. Then I washed his mouth and scraped his tongue; and got abundance of corruption out of the same.... Then he desired to drink; I dissolved some of the confection in water and gave him thereof. Within an hour, this wrought a great alteration in him; in the eyes of all that beheld him."

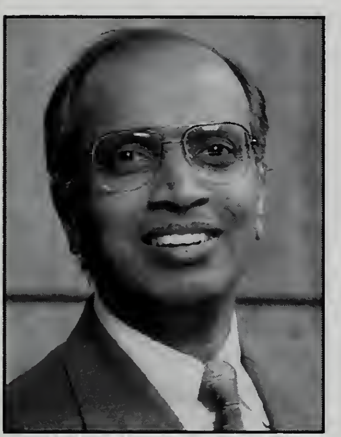
Winslow then sent back to the colony for chicken broth and such physic as the surgeon "durst" administer.

I do not wish to leave the impression that the simple nursing of Winslow, which was good medicine and was mingled, no doubt, with a fervent prayer, was wholly representative of the practices of the period. The colonial doctor, especially of the 18th century, had a great fondness for the lancet and bled his patients on the slightest provocation. He was an adept at concocting shotgun prescriptions and he sometimes had a grim confidence in boiled or calcined toads, decoctions of sow bugs and nauseating and horrible—but impressive—remedies in general. Later also there came to the towns some practitioners of less sterling character than the early doctors, a feature which contributed toward the needs of standards in ethics, education, and licensing and was one of the contributing forces toward the founding of medical societies.

In my philosophizing on the characteristics of our early physicians, I have left myself no time for the men themselves. You will find them listed in the scholarly Connecticut medical bibliography by Dr. Steiner in the recent publication of this society, *The Heritage of Connecticut Medicine*. Nor have I touched upon the gradual changes that came in the medicine of Connecticut with the growth of European medicine in the 18th century, with the collection of books in this country, with the growth of cities here, with the greater culture of our doctors as life became less primitive, but more complex. And I have said nothing of the contribution of our doctors to that war which marked the close of the colonial period. I have, as I said in the beginning, tried only to define something of the early forces which have left indelibly their mark upon the medicine of Connecticut.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

Doctors' Worth



It has long been my belief that we have not informed the public about our own values and our worth. Are we paid too much for our services or too little? The public's perception of doctors as overpaid professionals is wrong. A minority of doctors making a lot of money should not be considered the norm or the average. On this subject one brave man has come forward and written the truth in his column. He is the syndicated columnist Mr. Mike Royko. We have gotten permission to reprint the article. I hope it will serve as a useful weapon for our colleagues to counter the criticism. Here it is!

Sultan Ahamed, M.D.
President

When Polling the Stupid, Results are Predictable

MIKE ROYKO

On a stupidity scale, a recent poll about doctors' earnings is right up there. It almost scored a perfect brain-dead 10.

It was commissioned by some whiny consumer's group called Families USA.

The poll tells us that the majority of Americans believe that doctors make too much money.

The pollsters also asked what a fair income would be for physicians. Those polled said, oh, about $80,000 a year would be OK.

How generous. How sporting. How stupid.

Why is this poll stupid? Because it is based on resentment and envy, two emotions that ran hot during the political campaign and are still simmering.

You could conduct the same kind of poll about any group that earns $100,000-plus and get the same results. Since the majority of Americans don't make those bucks, they assume that those who do are stealing it from them.

Maybe the Berlin Wall came down, but don't kid yourself, Karl Marx lives.

It's also stupid because it didn't ask key questions, such as: Do you know how much education and training it takes to become a physician?

If those polled said, no, they didn't know, then they should have been disqualified. If they gave the wrong answers, they should have been dropped. What good are their views on how much a doctor should earn if they don't know what it takes to become a doctor?

Or maybe a question should have been phrased this way: "How much should a person earn if he or she must (a) get excellent grades and a fine educational foundation in high school in order to (b) be accepted by a good college and spend four years taking courses heavy in math, physics, chemistry, and other lab work and maintain a 3.5 average or better, and (c) spend four more years of grinding study in medical school, with the third and fourth years in clinical training, working 80 to 100 hours a week, and (d) spend another year as a low-pay, hard-work intern, and (e) put in another three to 10 years of postgraduate training, depending on your specialty, and (f) maybe wind up $100,000 in debt after medical school, and (g) then work an average of 60 hours a week, with many family doctors putting in 70 hours or more until they retire or fall over?"

As you have probably guessed by now, I have considerably more respect for doctors than does the law firm of Clinton and Clinton, and all the lawyers and insurance executives they have called together to remake America's health care.

Based on what doctors contribute to society, they are far more useful than the power-happy, ego-tripping, program-spewing, social tinkerers who will probably give us a medical plan that is to health what Clinton's first budget is to frugality.

(continued on next page)

Reprinted by permission: *Tribune Media Services.*

(continued from previous page—The President's Page)

But propaganda works. And, as the stupid poll indicates, many Americans wrongly believe that profiteering doctors are the major cause of high medical costs.

Of course doctors are well compensated. They should be. Americans now live longer than ever. But who is responsible for our longevity—lawyers, Congress, or the guy flipping burgers in a McDonald's?

And the doctors prolong our lives despite our having become a nation of self-indulgent, lard-butted, TV-gaping couch cabbages.

Ah, that is not something you heard President Clinton or Super Spouse talk about during the campaign or since. But instead of trying to turn the medical profession into a villain, they might have been more honest if they had said:

"Let us talk about medical care and one of the biggest problems we have. That problem is you, my fellow American. Yes, you, eating too much and eating the wrong foods; many of you guzzling too much hooch; still puffing away at $2.50 a pack; getting your daily exercise by lumbering from the fridge to the microwave to the couch; doing dope and bringing crack babies into the world; filling the big city emergency rooms with gunshot victims; engaging in unsafe sex and catching a deadly disease while blaming the world for not finding an instant cure."

"You and your habits, not the doctors, are the single biggest health problem in this country. If anything, it is amazing that the docs keep you alive as long as they do. In fact, I don't understand how they can stand looking at your blubbery bods all day."

"So as your president, I call upon you to stop whining and start living cleanly. Now I must go get myself a triple cheesy-greasy with double fries. Do as I say, not as I do."

But for those who truly believe that doctors are overpaid, there is another solution: Don't use them.

That's right. You don't feel well? Then try one of those spine poppers, needle twirlers, or have Rev. Bubba lay his hands upon your head and declare you fit.

Or there is the do-it-yourself approach. You have chest pains? Then sit in front of a mirror, make a slit here, a slit there, and pop in a couple of valves.

You're going to have a kid? Why throw your money at that overpaid sawbones, so he can buy a better car and a bigger house than you will ever have (while paying more in taxes and malpractice insurance than you will ever earn)?

Just have the kid the old-fashioned way. Squat and do it. And if it survives, you can go to the library and find a book on how to give it its shots.

By the way, has anyone ever done a poll on how much pollsters should earn?

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

The Emerging Legal Consensus About End of Life Decisions

JOSEPH M. HEALEY, J.D.

THE 18-year period since the *Quinlan* case has produced a series of judicial decisions, statutes, and administrative regulations helping to clarify the legal consensus about end of life decisions including: 1) the nature of a doctor's duty to provide or to continue treatment; 2) when such a duty exists or continues; and 3) how such a duty should be carried out. In a recent article,[1] Alan Meisel, author of a leading treatise on legal issues at the end of life,[2] has identified eight points of this consensus that have emerged during this period. His description of the consensus helps us to appreciate both the settled and unsettled areas of health policy and to recognize remaining areas of continuing controversy.

Meisel acknowledges that his points of consensus do not reflect unanimous agreement, and he recognizes the existence of some dissenting voices. But those appear to be in the minority. His eight points are:

1. Competent patients have a common-law and constitutional right to refuse treatment.

2. Incompetent patients have the same rights as competent patients; however, the manner in which these rights are exercised is, of necessity, different.

3. No right is absolute, and limitations are imposed on the right to refuse treatment by societal interests.

4. The decision-making process should generally occur in the clinical setting without recourse to the courts.

5. In making decisions for incompetent patients, surrogate decision-makers should apply, in descending order of preference, the subjective standard, the substituted judgment standard, and the best interests standard.

6. In ascertaining an incompetent patient's preferences, the attending physician and surrogate may rely on a patient's "advance directive."

7. Artificial nutrition and hydration is a medical treatment and may be withheld or withdrawn under the same conditions as any other form of medical treatment.

8. Active euthanasia and assisted suicide are morally and legally distinct from forgoing life-sustaining treatment. [1, p. 315]

The first seven points reflect principles currently applied in most jurisdictions governing the withholding or withdrawing of beneficial or nonbeneficial treatment. The eighth point remains controversial with respect to two types of cases: 1) acts producing unintended but foreseeable consequences (the principle of "double effect"), and 2) acts intending the death of the patient. Four types of cases highlight the difficulty in achieving consensus on this point:

1. The use of pain killers in increasing doses, risking the death of the patient;[3]
2. The prescription of barbiturates for a patient with advanced acute myelomonocytic leukemia;
3. An injection of morphine sulfate into a patient with advanced ovarian cancer;[5]
4. Facilitation of a patient's use of a suicide machine.[6]

The case presented by Jonsen and Lo is a good example of type 1.[3] The patient was a 60-year-old machinist, hospitalized for metastatic cancer. Two years previously, carcinoma of the colon was detected and the patient underwent surgical resection and adjuvant chemotherapy. During the prior month, the patient's symptoms had included abdominal and lower back pain, weight loss, and weakness. Progressive vomiting and urinary retention prompted an admission to the hospital through the emergency room. Examination at admission revealed a hard pelvic mass. Further studies confirmed a solid pelvic mass and bilateral hydronephrosis. Recurrent metastatic carcinoma was diagnosed. Knowing the impossibility of successful treatment, the patient and his family refused any further treatment other than the use of pain mediction (including morphine), nasogastric suction, and intravenous fluids. The patient's mind was described as clear as he spoke openly and affectionately about his approaching death.

(continued on next page)

JOSEPH M. HEALEY. J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

Correspondents: Joseph M. Healey, J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington, CT 06030-1910.

(continued from previous page)

Does the use of therapeutically appropriate levels of mediciation to achieve the goal of pain relief justify taking the risk that an undesired outcome might eventuate? Would the use of a significantly higher does of pain medication be justified? These issues will be explored in next month's column.

REFERENCES

1. Meisel A: The legal consensus about forgoing life-sustaining treatment: Its status and its prospects. *Kennedy Inst Ethics J* 1992; 2:309-45.
2. Meisel A: *The Right to Die*. New York, NY: John Wiley & Sons; 1989.
3. Lo B, Jonsen A: Ethical decisions in the care of a patient terminally ill with metastatic cancer. *Ann Intern Med* 1980; 92:107-11.
4. Quill T: Death and dignity: A case of individualized decision making. *N Engl J Med* 1991; 324:691-4.
5. Anonymous: It's over, Debbie. *JAMA* 1988; 259:272.
6. Annas G: Killing machines. Hastings Cent Rep 1991; 21:33-5.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

CALL FOR ABSTRACTS

1st Annual Scientific Assembly
of the
Connecticut College of Emergency Physicians
November 3, 1993

The Research Committee of the Connecticut College of Emergency Physicians announces the first annual scientific assembly. Robert Schwartz, M.D., is accepting abstracts for review for oral and poster presentation. Authors are urged to submit original work in all aspects of Emergency Medicine including cerebral resuscitation, pediatrics, cardiopulmonary resuscitation, toxicology, administration, trauma, EMS, shock, basic sciences, infectious disease, injury prevention and methodology.

Other topics are welcome.

Abstracts must be postmarked by August 1, 1993.

Requests for abstract guidelines and abstract forms should be to:

Robert Schwartz, M.D., M.P.H.
Department of Emergency Medicine/Trauma
Hartford Hospital
80 Seymour Street
Hartford, CT 06115
(203) 524-3112

Geriatrics: Defining a New Specialty

ROBERT U. MASSEY, M.D.

> *In as much as I no longer cling so hard to the good things of life when I begin to lose the use and pleasure of them, I come to view death with much less frightened eyes.*
>
> —Montaigne[1]

IN geriatrics we have seen the birth and growth of a major medical specialty that defines itself in part by the age of its patients. Like its analogue pediatrics, whose practitioners in the early decades of this century declared their patients to be more than little adults, the protagonists of geriatrics argue with justification that their patients are not simply older adults. Moreover, just as infant feeding is hardly the province of the internist, so urinary incontinence may be brushed aside by the generalist as one of those inconveniences old men and women must endure. In but a score of years the small company of geriatricians has acquired new information about old problems that before had been accepted as inevitable, intractable accompaniments of aging. Furthermore, they have, like pediatricians, helped to shape public policy in support of their patients. They have defined and justified a body of knowledge that cuts across and enhances almost all of the older specialties, and have made long-term care a worthy academic discipline.

> Withering is nature's preparation for death, for the one who dies and for those who look upon him. We may wish to flee from it, perhaps, or seek to cover it over, but we must be cognizant of the costs of doing so.[2]

Because all of us have known octogenarians in full possession of their powers, we should have questioned our predilection for labeling illnesses that occur after Medicare as simply manifestations of senility. Kindly, most of us have said more than once, "Remember, you're not as young as you once were!" Or irritably, "What do you expect at your age?" Doctors, because they pass their days among the sick come to exaggerate their numbers, and, because many of the sick are old, come to believe that most elderly are frail, sick, and demented. I often forgot that my uncle, who lived in excellent health past 90, survived being gored by a bull at 92, and decided to give up smoking for good while recovering from the surgery to repair his small bowel. Last summer a close friend, age 87, fell off a roof, fractured his pelvis and three ribs, and was back working in two weeks. Everett Koop, then surgeon general, described a meeting with his old surgical chief, Jonathan Rhoades, by then 81, who declared he was fine, but that his practice was "falling off a little bit."[3] Heinrich Schütz, the early baroque composer, wrote his "Christmas Oratorio" at age 80 and Giovanni Battista Morgagni published his great work on anatomic pathology, *De sedibus et causis morborum* when he was 79.

Yet there are diseases and disabilities that are frequent in old age, and some that are peculiar to the elderly. To deny that aging changes organs and tissues, diminishes physiologic functions, and that withering goes on inexorably to death is to deny mortality and the benign order of nature. Too much busy intervening in this process will exact costs that we should never incur, yet our expensive, ingenious, specialist-driven technology is pushing us to do just that.

But geriatrics is not about such things; is not, if I understand it, seeking to extend our life span beyond that devised by nature. Rather, as the articles in this issue of *Connecticut Medicine* illustrate, geriatrics seeks to learn the ways of successful aging and to offer beneficent, simple, and reliable guides along the road that most of us, willy-nilly, will travel. The voice that is missing, however, is the voice of the elderly themselves. With their advocacy of autonomy, the geriatricians will help them regain it, and then listen to all that those of them who "view death with much less frightened eyes" may have to say about growing old!

REFERENCES

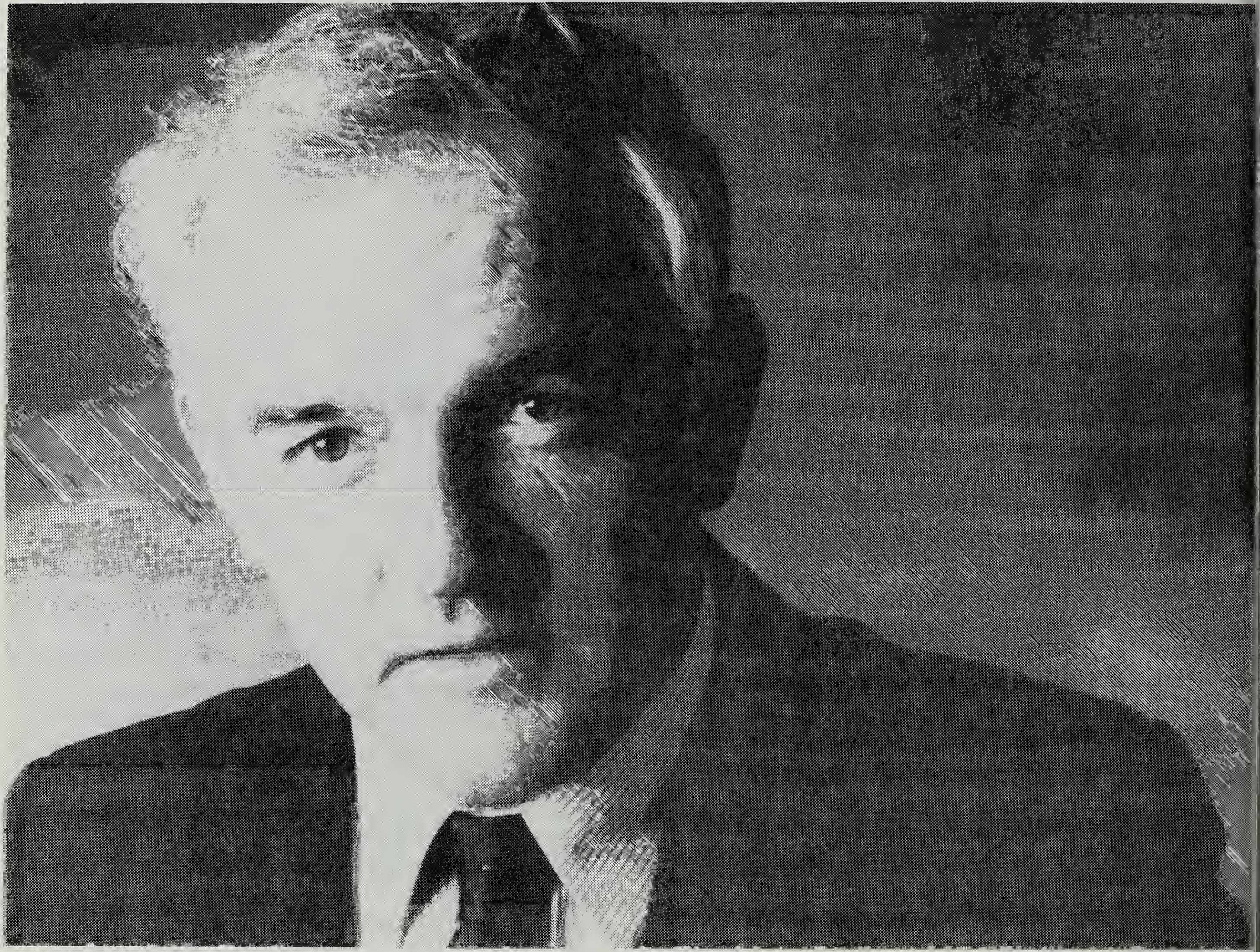
1. From Montaigne, "That to Philosophize Is to Learn to Die," quoted in Kass LR: *Toward a More Natural Science*, New York: The Free Press; 1985:307.
2. Kass LR: Mortality and morality: The virtues of finitude, *Toward a More Natural Science*. New York: The Free Press; 1985:299-317.
3. Koop CE: *Koop: The Memoirs of America's Family Doctor*. New York: Random House; 1991:63.

ROBERT U. MASSEY, M.D., Professor Emeritus, Division of Humanistic Studies, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

"Filling out insurance forms gives my patients a pain in the neck."

—Dr. Patrick J. Carolan, Orthopedics, Bridgeport, CT



"Good medical care was once as easy as seeing your doctor. Complications set in. Forms to fill out. Deductibles. Hidden costs. Partial coverage. Non-medical intrusions into the patient-physician relationship.

"Doctors decided to do something about it. Through our State Medical Society, we first created **CSMS-IPA,** Connecticut's only *statewide* IPA. Then we launched **M.D. Health Plan,** based on a simple principle: The best medicine for *patients* is practiced by *physicians*— not insurance companies, not benefit managers, not cost analysts.

"It worked! Now the fastest growing health plan in Connecticut and the choice of over 68,000 patients, our company is 100% owned and 100% directed by practicing physicians. Why not?"

Patrick Carolan, M.D.,

M.D. HEALTH PLAN

6 DEVINE STREET • NORTH HAVEN, CT 06473 • (203) 776-5759 • 1-800-345-9272

This Month's Reading in Review

TIMOTHY B. NORBECK

- "Back in the 1970s, we thought HMOs were going to solve all our problems. They didn't. And now we hear the same thing about managed competition. It won't."—Stuart Altman, chairman of the Prospective Payment Assessment Commission.

- Another reason why the State of Connecticut ranks third in the country in per capita health care expenditures is that it spends more per capita on each Medicaid patient ($5,994) than any other state....New York was second at $5,577....The Medicaid program's federal share for 1992 was $65.9 billion, while the states' contributions were $48.6 billion....The federal share of expenditures is based on each state's per capita income, with an established maximum 83% contribution and 50% minimum.

- Mississippi, with a per capita income of $8,088, had 79.9% of its Medicaid expenditures reimbursed by the federal government in 1991, while Connecticut, with a per capita income of $16,094, was reimbursed at a rate of only 50%.

- According to a report published by the National Federation of Independent Business Foundation, 44% of workers now in companies without health insurance coverage would have had some form of coverage if state level mandates were eliminated....The National Center for Policy Analysis estimates that 25% of the people who do not have health insurance have been priced out of the market by the costs of mandated coverage.

- According to Mandated Benefits Manual, states have passed 1,035 benefit mandates although the Blues' association places that figure closer to 950....Two-thirds of the Blues' plans estimated that the mandates increased their costs 10% to 21%....The most common provider mandates are chiropractors (41 states), psychologists (39) and optometrists (35), while the most frequently mandated services are mammography screening (43 states), alcoholism treatment (42) and treatment for mental health and treatment of drug abuse (31).

- According to *Health Care Financing*, the average length of a stay in a U.S. hospital was 9.1 days in 1990....In Japan, the average was 50.5 days....The average hospital stay in Great Britain was 14.5 days, in Germany 16.2 days, and Canada 13.9 days.

- According to a NIH study, more than one in four adults (28%) in the U.S. suffer from a mental disorder each year and only 28% of them seek professional help.

- Sky Chef, the airline food service company based in Arlington, Texas, hired benefits consultants Hewitt Associates to analyze their health data over 1990 and 1991 in which the firm's health care costs increased by 40% annually....The results were significant....They found that 63% of all claim dollars for 8,000 employees were related to lifestyle....A redesigned health plan that motivates employees to change health habits and seek care earlier has cut health costs by 7.5% in the first six months.

- The portion of out-of-pocket expenses has shrunk during the past 20 years, from almost 46% of overall health costs to 19% of costs by 1991.

- According to a newly released A. Foster Higgins survey, some managed care programs in Houston, Texas, cost more than traditional indemnity plans....The average cost of health plans in Houston per employee: indemnity plans, $3,630; HMO, $3,580; PPO, $4,100; and POS, $5,650.

- "This man is costing my health plan $5,000 a day! I demand that he die with dignity." (Homer Simpson's employer Mr. Burns, as he burst into Homer's hospital room after Homer had fallen into a coma). "The Simpsons," FOX (1 April 1993).

- An Alan Guttmacher Institute study indicates that more than one in five Americans—56 million people—are infected with a viral sexually transmitted disease.

- *Only in America:* Within an hour of the blast at the World Trade Center on 26 February, one survivor escaped and managed to reach a pay phone. His first call was made to his lawyer to see what damages he could seek....Within one hour of the blast, one New York law firm received "30 to 40 calls" from people who were near the World Trade Center at the time.

TIMOTHY B. NORBECK, Executive Director, Connecticut State Medical Society.

Hospital Medical Staff Section
21st Assembly Meeting
Chicago Marriott Hotel
June 10-14, 1993

Reform of the American health care delivery system is the issue of the 90s. The public and political debate over changing our health care delivery system is intense. Significant reform legislation has already been passed in several states. To help you lead your organization in a time of major change, three educational programs will be part of this HMSS Assembly Meeting.

Negotiations and Conflict Resolution

Gain new skills to effectively provide leadership in negotiating and resolving conflicts that arise in the process of health care delivery. Learn to frame negotiation in the best interests of all parties, to listen actively, to use positional bargaining and avoid surprises.

Review and Analysis of President Clinton's Health Care Reform Plan

President Clinton is expected to release his new health care reform plan in May. Participants in this session will hear a full review and analysis of the plan from leading experts in the medical field.

Challenge and Change in the Managed Care Environment

Develop a comprehensive understanding of the current environment in health care highlighted by a discussion of the impact of President Clinton's plan on health care delivery system reform. Discover steps physicians can take to deal with the realities of managed care.

Don't miss this opportunity to acquire leadership skills to help your physician community succeed!

For more information, please call
312 464-4754 or 464-4761.

Hospital
Medical
Staff
Section

American Medical Association
Physicians dedicated to the health of America

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

The Patient Care Evaluation System: Patients' Perception of Anesthetic Care

LINDA M. PREBLE, R.N., M.P.S, LAWRENCE PERLSTEIN, M.B.A.,
LORI KATSOFF-SEIDMAN, B.A., THERESA Z. O'CONNOR, M.P.H.,
AND PAUL G. BARASH , M.D.

ABSTRACT—Our department's Patient Care Evaluation Survey was developed to measure the patients' perception of the care they received by our anesthesia caregivers (attending physicians, residents, and certified registered nurse anesthetists [CRNAs]), and identify those areas of patient contact which might require improvement in the delivery of anesthesia. The survey instrument (a personal letter sent by the chairman), is mailed to randomly selected patients two weeks following discharge from the hospital or ambulatory surgery center. Patients rated the anesthesia care they received from one (poor) to 10 (excellent) in three areas: the preoperative visit, the postoperative visit, and overall satisfaction.

From October 1986 to October 1991, a total of 2,374 (n=2,374) questionnaires representing a random sample of 99,964 anesthetics, were sent to patients. Fifty-four percent or 1,291/2,374 patients returned the survey. A high degree of patient satisfaction is seen for all categories: the preoperative visit, (mean score of 9.17±1.6); the postoperative visit (8.33±2.7); and overall satisfaction (9.15±1.7). Of the responses returned, 0.9% of the patients surveyed rated their interaction with the department as unsatisfactory in all three areas; 2.6% in two areas; 10.8% in at least one area.

The Patient Care Evaluation system has served as an important means of feedback for medical, educational, and administrative goals of the department.

Introduction

PATIENT satisfaction is important for maintaining the health of the consumer and for positive public image of health care providers. It is consistent with the trend toward holding the health professional accountable to the patient.[1] Most physicians are able to establish patient relationships over a period of time. Therefore, determining patient satisfaction is not as difficult for the primary physician as it is for the anesthesiologist. The anesthesiologist usually meets the patient one to 24 hours prior to surgery and does not have an opportunity to establish a continuing relationship. The ability of the anesthesiologist to establish a reasonable relationship in this short time frame is imperative and crucial in providing safe patient care. However, inadequate communication and/or interview skills, a patient's psychosocial concerns, residual effects of anesthetic agents, or postoperative sedation and analgesia may serve to impair interactions between the anesthesiologist and patient during the *perioperative* period. To date, there has been little published data regarding patients' satisfaction with their anesthetic care. As part of our department's Quality Assurance process, we developed the Patient Care Evaluation System to measure the patients' perception of the care they received, evaluate the performance of our anesthesia caregivers (attendings, residents, and CRNAs), and identify those areas of patient contact that might require improvement.

Methods

Fourteen days following discharge from the ambulatory surgery center or hospital, randomly selected patients (one of every 40 patients anesthetized by members

---

LINDA M. PREBLE, R.N., M.P.S., Clinical Administrator, Pain Management Services; LAWRENCE PERLSTEIN, M.B.A., Administrator; LORI KATSOFF-SEIDMAN, B.A., Administrative Associate; THERESA Z. O'CONNOR, M.P.H., Biostatistician; PAUL G. BARASH , M.D., Professor and Chairman, Department of Anesthesiology, Yale University School of Medicine, Yale-New Haven Hospital, New Haven.

Address correspondence: Paul G. Barash, M.D., Department of Anesthesiology, Yale University School of Medicine, P.O. Box 3333, 333 Cedar Street, New Haven, Connecticut 06510. Fax: (203) 785-6664; Phone: (203) 785-2802.

of the Department of Anesthesiology) are sent a personal letter from the chairman of the department. Those patients with risk management issues, under the age of 18, or cases in which a death occurred were excluded. The Patient Satisfaction Survey asks the following questions:

Q1: Do you feel that the anesthesia preoperative examination and evaluation allowed time for you to review your concerns regarding anesthesia? [Score 1 (poor) - 10 (excellent)].

Q2: Were you seen postoperatively and given an adequate opportunity to discuss your anesthetic experience? (Score 1-10).

Q3: Overall, how would you rate the anesthesia care you received? (Score 1-10).

In addition to the subjective score for each of these questions, written comments are also solicited from the patient.

Following return of the survey, Quality Assurance data and patient responses are coded, collated with demographic information (age, sex, primary anesthetic technique, procedure, and date of service), entered into a database, and are then statistically analyzed. Each of the patient's responses for questions one to three are entered into one of two response groups: ≥ 5 or < 5. The < 5 response group is considered unsatisfactory and, in these particular cases, each chart is individually reviewed by the chairman. Specific concerns raised by patient responses are addressed with a follow-up letter and/or communication by the anesthesia care team who managed the patient. Data gathered from the questionnaires are expressed as either percent or mean ± SD.

Data were analyzed by the Student's *t* test for unpaired data, and χ^2 or Fisher's exact test where appropriate, $P < 0.05$ is considered statistically significant.

Results

From October 1986 to October 1991, a total of 2,374 (n=2,374) questionnaires representing a random sample of 99,964 anesthetics, were sent to patients. Fifty-four percent or 1,291 patients returned the survey. To investigate whether the nonresponder patient population pool was similar to responders, we compared the two groups with respect to age, sex, surgical procedure, and type of anesthetic. The groups did not differ by any of these demographic variables (*P*=NS).

Table 1 shows a high degree of patient satisfaction for all categories contained in the survey: 11/1,291(0.9%) rated their interaction with the department as unsatisfactory (score less that 5) in all three areas addressed by the survey; 33/1,291(2.6%) rated their interaction with the department as unsatisfactory in two areas; and 140/1,291(10.8%) rated their interaction with the department as unsatisfactory in only one area. The postoperative visit, which has consistently received the lowest score, was identified by the survey as an area of patient concern.

Table 1.—Survey Responses

	N= 1291 X+ SD
Preoperative Evaluation	9.17 ± 1.6
Postoperative Visit	8.33 ± 2.7
Overall Anesthetic Care	9.15 ± 1.7

Under usual circumstances, the department's Quality Assurance statistics demonstrate that over 91% of patients receive a postoperative visit. In reviewing patient response scores and comments, a total of 161 (n=161) patients responded that they did not have a postoperative visit. A retrospective chart review revealed that only 33 patients (33/1,291), who said they were not seen in the postoperative period were, in actuality, not seen. The remaining 128 patients were seen and were unaware of the postoperative visit. In fact, 99/128 were visited at least once and 29/128 were seen at least twice. When the demographics of the 33 survey patients who were not seen postoperatively were compared to the remainder of the study population, no significant differences were observed with respect to age, sex, anesthetic technique, or surgical procedure (*P*=NS).

Examining departmental data for the course of the study demonstrates that the percentage of patients who do not remember being visited in the postoperative period is declining (11.9% in the first 32 months *vs* 9.2% in the latter 34 months; *P*=0.16).

Discussion

Two 1978 studies conducted in Great Britain[2,3] regarding patients' views of the anesthesiologist concluded that patients had insufficient and imprecise knowledge about the anesthesiologist's role and did not recognize that they are medically qualified personnel. A study undertaken in 1991 by Shevde et al[4] concluded that the public's awareness of the anesthesia care team had changed very little.

The Patient Care Evaluation System measures the patient's perception of the anesthetic care and evaluates the anesthesia care team's interaction with the patient in an effort to improve departmental performance. It differs from other surveys as it is patient-centered rather than physician-oriented. Most quality assurance surveys focus on the appropriateness of care that is given by the provider. In addition, surveys sent to patients by hospitals or practice plans attempt to evaluate overall performance regarding care, setting, atmosphere, etc. There is less of a focus by the patient on specific anesthesia care issues when large general surveys, rather than specific departmental ones, are utilized.

The subjective ratings and written responses in our survey suggest that patients are becoming increasingly aware of the contributions of the anesthesiologist in perioperative care. For example, one patient responded:

> I am happy to be alive and recovering well. If that is your goal, as it is mine, then you may rate your department as excellent. If your goal is to assist the patient to adjust to the effects, postoperatively, of general anesthesia, to be well informed, and to be completely satisfied, then you'd have to give yourselves a lower rating. If a member of your staff had spent a few minutes after I regained consciousness, or placed a paper in my hands explaining the common after-effects of the type of anesthesia used, I'd have had an understanding of what to expect, that what I was experiencing had a cause, and that the symptoms would pass. I consider this information my *right.*

Furthermore, the system has also served as an important means of feedback for medical, educational, and administrative goals of the department. It has also answered unresolved questions which some patients had concerning their anesthetic care. Because of the residual effects of anesthetics and postoperative analgesics, some patients do not remember being visited postoperatively and, therefore, cannot ask relevant questions. For example, one patient commented:

> I am told that I was seen postoperatively, but I wasn't clear-headed enough to even remember. I was told that I required more general anesthesia than usual and I'm not sure of it's effect on me and the baby, or why. Perhaps the anesthesiologist should have come to see me when I was more able to concentrate—when the anesthesia was truly out of me, as well as after the demerol PCA pump was removed.

This patient received a follow-up letter from the chairman and anesthesia care givers regarding her anesthetic concerns. The analysis of survey data identified the postoperative visit as an area in which changes should be made. Examining departmental data over the last 64 months demonstrates that the percentage of patients who do not remember being visited in the postoperative period is declining. However, further changes are still necessary. Changes that could be instituted include handing out patient information cards that include the type of anesthesia, residual/side effects to expect, and the name of the anesthesiologist (for future patient reference) or visiting the patient on the second or third postoperative day, rather than the first.

Secondly, our Patient Care Evaluation System gave important educational feedback to members of the anesthesia team who managed the patient. For example:

> The anesthesiologist, who was very friendly and outgoing, explained everything so I understood completely and put me at ease before my surgery.

Thus positive comments may serve to reinforce established clinical practice. Alternatively, clinical practice patterns may require change based on comments such as the response from this nurse undergoing emergency gastroenterology surgery, who stated:

> I felt as though I was being choked to death just prior to being put to sleep.

In reviewing the perioperative course of events from this patient's chart and in discussing the case with the anesthesia care team, it was noted that a Sellick's maneuver had been performed prior to the induction of anesthesia.

The feedback given to our anesthesia care teams from the Patient Care Evaluation System has served to heighten the department's awareness of how the patient views anesthesia as well as to what extent the anesthesiologist needs to concentrate on improving communication in particular areas of patient care, interests, concerns, or questioning. The patient comment above demonstrates the importance of communicating with the patient and explaining what is being done in order to relieve the anxiety and misconceptions.

Shevde and colleagues believe that the anesthesiologist should specifically inform patients about their role in monitoring vital signs and maintaining normal hemodynamics, in addition to addressing the patient's concerns regarding waking up and experiencing pain.[4] Furthermore, Bertrakis demonstrated that interview styles in which physicians adopted a more affiliative and patient-centered communication were related to higher patient satisfaction than when physicians displayed a more domineering attitude.[5] He further stated that it is appropriate to continue to study how physicians' communication behaviors influence patient satisfaction. The intelligent presentation of information with adequate time for discussion of the patient's concerns and fears go far toward strengthening a patients's insight into anesthesia care and, in fact, may enhance anesthesiologist-patient relationships and communication. In general, the greater the friendliness and solidarity expressed by the physician, the more satisfied was the patient.[6]

Finally, the survey permitted the departmental chairman to have a broad overview of the anesthetic care being administered by members of his department. For example, the patient's response scores and written comments concerning individual caregivers helped to determine if any *one individual* had an excessive number of negative comments or consistently low scores. Because of the high degree of patient satisfaction, we have found no one individual with a significant number of negative comments regarding patient care, communication, or interaction. This may be due to the immediate follow-up of patient care issues by the chairman, as well as rapid feedback to members of the anesthesia care team in order to modify patient-caregiver interactions as deemed necessary.

In conclusion, our Patient Care Evaluation System measures the satisfaction of anesthesia care from the patient's perspective to determine areas that need to be addressed and to identify interpersonal problems that may exist. The practice of medicine in today's society mandates that every health-care provider, including anesthesiologists, monitor the appropriateness and effectiveness of their care. Our department has undertaken this challenge through the development of the Patient Care Evaluation System.

REFERENCES

1. Merkel WT: Physician perception of patient satisfaction: Do doctors know which patients are satisfied? *Medical Care* 1984; 22:453-9.
2. Keep PJ: Forum: As others see us. The patient's view of the anesthetist. *Anesthesiology* 1978; 33:43-5.
3. Herman CR: An appraisal of the anesthetist-patient relationship. *Anesthesiology* 1978; 33:45-8.
4. Shevde K, Panagopoulos G: A survey of 800 patients' knowledge, attitudes, and concerns regarding anesthesia. *Anesth Analg* 1991;73:190-8.
5. Bertakis KD, Roster D, Putnam SM: The relationship of physician medical interview style to patient satisfaction. *J Fam Pract* 1991; 32:175-81.
6. Constock LM, Hooper EM, Goodwin JM, Goodwin JS: Physician behaviors that correlate with patient satisfaction. *J Med Ed* 1982; 57:105-12.

YOCON®
YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.
Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.
Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.
Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.
Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.
Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]
How Supplied: Oral tablets of YOCON® 1/12 gr. 5.4mg in bottles of 100's NDC 53159-001-01, 1000's NDC 53159-001-10 and Blister-Paks of 30's NDC 53159-001-30
References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85

Available at pharmacies nationwide
PALISADES PHARMACEUTICALS, INC.
219 County Road
Tenafly, New Jersey 07670
(201) 569-8502
(800) 237-9083

HEALTH SCIENCES LIBRARY
BALTIMORE

Introducing Physicians to Case Management in Psychiatry

ALBERT N. BROWNE-MAYERS, M.D.

ABSTRACT—All physicians sooner or later find themselves in contact with a case manager whose job is to help the noninstitutionalized psychiatric patient who is chronically ill. This paper is an attempt to orient the physician about this phase of treatment which started not many years ago. The article gives a short history of the development of case management and demonstrates the various steps the case manager takes in working with a patient. These are, assessment of the problems, plans to meet the problems, linkage with agencies, monitoring the progress, advocacy, and evaluation. These activities may bring about improvement in the kind of patient frequently considered hopeless.

SINCE most chronically ill mental patients have been deinstitutionalized most physicians, especially those in primary care, sooner or later will be involved with such patients.

How does the physician come in contact with the patient? Usually the chronically mentally ill will have an illness that brings him to a family physician or primary care specialist in the community. The case manager may accompany the patient or may have made the appointment for the patient. Because of the problems often associated with chronic mental illness, the physician may seek or request some kind of intervention from the case manager to ensure that the patient will keep appointments and follow the care prescribed by the doctor. In such instances the case manager provides the link between the psychiatric clinic and the physician.

The psychiatric clinics in the northwestern part of the state are similar to most community mental health centers. They are staffed by psychiatrists, social workers, nurses, and psychologists. The chronically mentally ill are generally receiving psychotropic medication which is monitored by a psychiatrist once a month or, at times, more or less frequently. Whether at Torrington or at the Winsted Clinic the staff communicates readily either in formal or informal settings with the staff or the physician in the community. We believe our case managers have an advantage in that all of them have degrees in nursing and have served for years on the psychiatric inpatient department at Charlotte Hungerford Hospital. They have previously taken care of most of our chronic patients when they have decompensated and required a short-term rehospitalization.

The role of case manager* has been a relatively new development in Connecticut, having begun in 1986. The term case manager is not to be confused with the concepts of managed care or managed cases. Case manager is the usual term, but other names have been used such as assertive community treatment worker,[1] case coordinator.[2] Whether the term case manager or case coordinator is used, the service is the same. The object is to provide services for the chronically mentally ill, but the term chronically ill may have different definitions in different states. Usually such patients are those who require repeated hospitalizations or have served long periods of time in a state mental hospital. The number of chronically ill mental patients in the U.S. is about 22 million, so the total patient load nationally is a large one.[2] An important figure to contrast to this is that the number of patients in state institutions in the United States in 1955 was 565,000, but now has shrunk to less than 180,000.[3] Currently the number of beds allotted to our small area at the state hospital has shrunk to 25.

ALBERT N. BROWNE-MAYERS, M.D., Staff Psychiatrist, Charlotte Hungerford Hospital, Torrington.

*With appreciation for the work performed by the case management team at Charlotte Hungerford Hospital: Marion Muschell, B.S.N., R.N.C.—Aftercare Coordinator, Ruth Savanella, R.N., A. Brenda Sherman, R.N., B.S., Carole Finn, R.N., and Donna Plaskett, L.P.N.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Historically, case management had its roots starting in the early 20th century with the development of social work. Much later in the 1950s deinstitutionalization of psychiatric patients began. Deinstitutionalization was accelerated by the discovery of psychotropic drugs that could quickly calm severe psychotic symptoms. Unlocked doors in the mental hospitals speeded the process and the civil liberties movement provided momentum for a policy of deinstitutionalization.[4] Passage of the Medicaid laws and the extension of Social Security disability payments to the mentally ill during the 1960s and 1970s made it possible for many patients to reside in the community. This last step was warmly welcomed by the states because the expense was shifted from state to the federal treasury. With deinstitutionalization it was expected that the newly formed community mental health clinics would be able to take care of the patients who had been discharged from the hospital. The author remembers a 1957 conversation with Paul Hoch, M.D., who later became Commissioner of Mental Health in New York State under Governor Nelson Rockefeller, during which he remarked that not all the patients should be deinstitutionalized, but because of economic problems more and more would be deinstitutionalized. Even though many psychiatrists complained about the practice of rampant deinstitutionalization, their criticism had no significant effect. Eventually in 1976, the General Accounting Office (GAO) pointed out the disadvantages associated with deinstitutionalization: the mentally disabled were being dumped into neighborhoods which were lacking in adequate services, and community care was not fulfilling the void created by having the patients leave the state hospital.[5]

As a result of these criticisms the National Institutes of Mental Health (NIMH) launched a modest but potentially significant program entitled, *The Communities Support System* (CSS). CSS was designed to improve services for one particularly vulnerable population: adult psychiatric patients whose disabilities were severe and persistent but for whom long-term skilled or semiskilled nursing care was inappropriate.[6] In this report it was pointed out that there had been fragmentation and confusion of responsibility and lack of effective community organization and advocacy. Out of this was developed a more comprehensive program labelled *Community Support Program* (CSP).

CSP then began a demonstration program sponsored by the National Institutes of Mental Health in 1978.[7] Other forces which helped develop case management came out of the President's Commission on Mental Health in 1978 which led to the Mental Health System's Act of 1980.[8] Out of the welter of these developments the job of case manager began.

Case management is frequently presented as a process comprising multiple functions without specifying the persons or structure responsible for carrying them out.[8] According to Agranoff, the five basic functions of a case management system include: 1) assessment of client's needs, 2) development of a comprehensive service plan, 3) arranging for services to be delivered, 4) monitoring and assessing services delivered, and 5) evaluation and follow-up.[9] These five functions—assessment, planning, linking, monitoring, and evaluation appear in almost every description of a case management system, regardless of its context.[10]

So far the basic functions of a case manager have been outlined. What happens in Connecticut? Here the Department of Mental Health has established various regions; this report is derived mainly from Catchment Area #22 which comprises 18 towns in the northwest area of Connecticut, predominantly semirural, with a population of approximately 97,300. The semirural quality has special significance that will be mentioned later. The state has seen to it that the case manager has a number of supporting agencies that the state provides. Among them are halfway houses, supervised apartments, and residential support with access to HUD, a federally supported program. Another federally funded program which has had several previous names is now called Bureau of Rehabilitation Services, in short BRS. Some of the facilities are growing and others disappearing. LARC which was a sheltered work shop is no longer in existence because of a shortage of funds. Privately run facilities of a nonprofit status are constantly changing in the northwest area. A parent company called Cooks Willow which was attached to Plymouth Hospital has given rise to two facilities. One is Cooks Life Skills which is funded through the Department of Human Services of the state. It is devoted towards enhancing simple daily life skills such as bodily hygiene, diet, budget keeping, psychosocial improvement. A different Cooks which is now part of Prime Time is copied from the program of Fountain House in New York City. It provides time-limited employment up to six months in duration with another program entitled Community Assistance Program which is not limited by a time factor. Vocational programs are also part of Prime Time. At the moment Prime Time is expanding the scope of its activity. Still another program called RESCUE deals with a variety of problems—part time employment especially for the retarded, jobs for retirees and the geriatric population, head start program, developmental delays of children either at home or in the class room.

As pointed out earlier these facilities seem to be in a state of flux and demands considerable knowledge and skill on the part of the case manager as to where to go for help for a particular patient. Finally, what happens with these helpful facilities in the northwest area may not occur elsewhere and considerable individuality may be expected comparing one community with another.

All patients are usually assessed before leaving a state psychiatric hospital or a local psychiatric inpatient service at a general hospital. At that time the case manager meets the patient and is oriented about the psychopathology, the psychodynamics, and the posthospital plans for the patient. The various strengths and weaknesses are considered. For instance, the patient may not have housing or may have no funds for support, and many linkages are therefore in order. The patient may need assistance in obtaining disability, social security, Medicaid, housing, or social and work rehabilitation. This aspect of linkage is closely allied to another phase of management advocacy. Advocacy helps the patient obtain entitlements or legal assistance. At this juncture the case manager serves as a liaison between the practicing physician and the psychiatric clinic and psychiatrist at the clinic. Because of the semirural community and rather stable population of doctors and patients, easy access between doctor and clinic usually occurs. Other important linkages may involve medical or surgical help.

After linkages are in place, the greatest effort is devoted to monitoring the plans. Sometimes the simplest procedures have to be supervised, such as complying with prescribed medication or taking a bath or wearing the proper clothes or cooking a balanced meal. Certain questions arise, such as, is the patient jeopardizing his ability to stay in the housing that has been obtained? The case manager has to see that the patient keeps appointments and arranges for transportation.

During the monitoring phase the patient may be seen frequently at home or while attending a supporting agency or in the psychiatric clinic for individual or group therapy sessions or for routine check-ups. The manager and the psychiatrist confer about the patient's progress. Many informal as well as scheduled team meetings are part of such monitoring; the monitoring is performed as long as the patient is on the active list of case management.

Advocacy for the patient may start at an early stage as was seen in the linkage phase. Occasionally, the patient needs help with obtaining social security or some other entitlements. For instance, one patient was getting his social security disability check but was paying almost as much for his health insurance. He did not realize that he could apply for Medicaid. Patients either do not know their entitlements or they make errors when they apply without the assistance of their case manager.

In view of the complex tasks that a case manager is asked to perform, what kind of a person is required? Intagliata[10] describes the job of case manager as follows: "integrator, expediter, broker, ombudsman, primary therapist, patient representative, personnel programmer coordinator, assistance's agent to name but a few, ... persons acting as case managers must be ready to play whatever role the situation may require—outreach worker, broker, advocate, teacher, community organizer, planner or administrator." "A variety of objectives have been associated with case management efforts. Perhaps the most fundamental of these is to enhance the continuity of care provided to clients."[11]

With such a job description could any one do the job? In our particular environment with many small towns, the director of the case managers believed that nurses, who had extensive experience in taking care of psychiatric patients, might do a better job.[12] Others have made a case for social workers.[2] When the nurses had known the patients previously because they had treated them on the inpatient service of the local hospital, the patients felt at ease with their managers. In view of the stable population the nurses often knew of the dysfunctional families in the area, and since some of the nurses had lived in the area most of their lives they were committed to helping a neighbor. Perhaps a more significant factor is that nurses are action oriented. As one nurse said, "When the emergency light flashes, the nurse runs to help."

Some nurses like the job because they can be their own boss most of the time. Being relatively independent has something to say for itself, even though the mentally disturbed patient in the back seat may stir up a certain amount of worry. Another advantage for nurses is that they have had experience with medically sick patients. They are used to such a challenge. Nurses who have worked on an inpatient service are also used to seeing the side effects of psychopharmacologic medicines and they can respond to this problem.

Working in a small town has another advantage too. The heads of agencies are people they have gotten to know over a period of years so that contacting them is easier and the patient's cause is not as likely to be lost as in a huge bureaucracy. Knowing the personnel in an agency helps the case manager to know what will work in a given case. Patients living in a semirural area are not at the mercy of inner city chaos; rural settings are more hospitable to the chronically mentally ill.[13]

Burn-out of the case manager is always a possibility. It takes only a few demanding patients to gobble up vast amounts of time. Burn-out is more likely to occur when the case manager has too many patients or lacks sufficient education or extensive experience for the job. In our semirural settings burn-out has occurred only once and that occurred when the case manager became over zealous in treating the alcoholics.

A case report will demonstrate those activities that are part of case management. In addition, this case demonstrates the fluctuational aspects of treatment:

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

The patient, now 71 years of age with a varying diagnosis of either schizoaffective or bipolar disorder with psychosis first came in contact with one of our case managers at the end of 1989. The patient was readmitted to the hospital after she had been picked up at a homeless shelter in deteriorated condition. For seven years prior to 1989 the patient had been in and out of several psychiatric services. Altogether she had about four psychiatric admissions with frequent visits to the emergency room at different hospitals. She was given different major and minor tranquilizers; none of them seemed to work over any length of time, probably because of noncompliance. Medically the patient was treated for a cardiac condition, left bundle branch block, elevated triglycerides, low thyroid function, and increased liver enzymes; only treatment for the cardiac condition remained consistent. The patient had seen many dentists because of her recurrent pain in the jaw, a symptom that subsided with tranquilizing medicines. No dental pathology was found. Careful work-ups for cerebral organicity were also negative.

Her first case manager took care of the patient from January to the beginning of March 1990. Her present case manager has looked after her up to the present. In the beginning it was clear that the patient was noncompliant with the medication. She needed housing and also some reconstructive social activity. By the end of March the patient had obtained housing, but because of noncompliance with medication, she was admitted to a psychiatric hospital once again for a month. In May when discharged, the patient was taking chlorpromazine 75 mg a day, desipramine 100 mg a day, and 40 mg of propanolol. Follow-up treatment became more intensive. She went to a facility for psychosocial rehabilitation five times a week where her medication was monitored. The case manager saw her three times a week and she also attended an aftercare group psychotherapy session twice a month. Progress and medication were reviewed monthly by a psychiatrist.

In September 1990 the patient overdosed on self-medication and was readmitted to the general hospital for detoxification for a few days. After discharge the patient was relatively stable with some fluctuations. In January 1991 she complained of not feeling well and was readmitted to a general hospital following a myocardial infarction. Also at that time the patient was treated for an overdose of salicylates. After consultation with an internist, medication was rearranged with higher doses of chlorpromazine. Desipramine was discontinued in view of her cardiac problems. Propanolol was also discontinued.

No matter how many times it was pointed out to the patient that whenever she stopped taking chlorpromazine her jaw pain returned, she never developed insight into the fact that she had to take chlorpromazine if she were to remain symptom free. Medication or not, the telephone wires kept up their activity—they were a constant source of hallucinatory material. The patient continued with monthly psychiatric check ups and aftercare group therapy.

Early in 1992 she quit her psychosocial rehabilitation group which resulted in the case manager monitoring her medications daily. She has remained relatively stable, shops for herself, attends church, and when the weather is warm, even goes fishing. When seen for monthly psychiatric check ups she is pleasant, amiable, and communicative, which is a sharp contrast to the elderly woman who was agitated, restless, and complaining about the delusional pain in her jaw and the noxious influence of the messages coming over the telephone wires. Not all patients are so difficult to stabilize but a small group of patients can absorb a tremendous amount of time from the case manager. On the other hand some of the chronic patients are able to make reasonably satisfactory adjustments outside the hospital so that the case manager gradually increases the time between follow-up contacts.

Originally it was hoped that it would be possible to determine whether caring for the patient after discharge was less costly than prolonged hospitalization, but this is no easy task because some of the costs are provided by the federal government, others by the state or local government, and others by private sources with frequent overlap. How to compare costs of institutional care with the present mode of case management seems almost impossible at this time. Those who have tried come up frequently with contradictory answers.

In conclusion, case management is usually an arduous task with frequent frustrations. Nevertheless our case managers find even their small successes give them courage to keep on going.*

REFERENCES

1. Witheridge TF: The assertive community treatment worker: An emergency role and its implication for professional training. *Hospital and Community Psychiatry* June 1989; 40:620-24.
2. Johnson PJ, Rubin A: Case management in mental health: A social work domain? *Social Worker* 1983; 28:49-54.
3. Johnson PJ: Case management, *The Field of Social Work*, 8th ed, Fink AE, Pfouts JH, Dobelstein AW, Sage Publications, Beverly Hills, 1985:269-96.
4. McLaughlin P: Case management: A short history and a case in point. *The Information Exchange* (newsletter) October 1987; 4:1-4.

---

*From the Committee on Rural Psychiatry of the Connecticut American Psychiatric Association composed of Nirmal Kathuria, M.D., Chairperson; Richard Kramer, M.D.; Donald Shapiro, M.D.; Albert Browne-Mayers, M.D.; Paul Sachs, M.D.; John Duers, M.D.; and Manage Nissanka, M.D.

5. Public Health Service Comments on the Draft General Accounting Office Report, Washington, DC. *Public Health Service* 19 October 1976.
6. Turner JC, Tenhoor WJ: The NIMH community support program. Pilot approach to a needed social reform. National Institute of Mental Health, *Schizophr Bull* 1978; 4:319-41.
7. Bond GR, Miller LD, Krumwied RD, Ward RS: Assertive case management in three CMHCs: A controlled study. *Hosp Community Psychiatry* 1988; 39:411-17.
8. The President's Commission of Mental Health, *Report to the President From the President's Commission on Mental Health*, 1978, Vol. 1, Washington, DC, Superintendent of Documents, U.S. Government Printing Office.
9. Argranoff R: Services integration, In: Anderson WF, Frieden BF, Murphy MJ, eds. *Managing Human Services* 1977, Washington, DC, International City Management.
10. Intagliata J: Improving the quality of community care for the chronically mentally disabled: The role of case management. *Schizophr Bull* 1982; 8:655-74.
11. Test MA, Stein LI: Use of special living arrangements: A mode for decision making. *Hosp Community Psychiatry* 1977; 28:608-17.
12. Lapierre ED, Padgett J, and Section Editors: Professionally speaking. *J Psychosoc Nur Ment Health Serv* 1992; 30:35-39.
13. Davies MA, Bromer EJ, Schulz SC, et al: Community adjustment of chronic schizoplirenic patients in urban and rural settings, *Hosp Community Psychiatry* 1989; 40:824-30.

"Ron's Rule—I give myself one week to meet new people and start having fun on a locum tenens assignment. It hasn't failed me yet."

Ron Richmond, MD, joined the CompHealth locum tenens medical staff when he completed his residency. He wanted to travel. He loves to meet people. A little time off sounded really good. And he thinks being exposed to different types of medical practice will serve him well when he returns to his hometown to establish a community health center.

A singer. A board-certified family practitioner. A soft-spoken New Yorker. Ron Richmond knows...

It's a great way to practice medicine

CompHealth

LOCUM TENENS

1-800-453-3030

Salt Lake City ■ Atlanta ■ Grand Rapids, Mich.

CALL FOR PAPERS

Members of the *Connecticut State Medical Society* reading papers before other organizations are requested to submit their papers to the JOURNAL for consideration by the Board of Editors for publication.

Authors preparing manuscripts for submission to *Connecticut Medicine* should consult **INFORMATION FOR AUTHORS**. This material may be obtained from the Journal office (also published in *Connecticut Medicine*). Adherence to the instructions will prevent delays both in acceptance and in publication. Manuscripts set with a computer word processing program should be submitted on disk along with hardcopy.

Please send them to:

Robert U. Massey, M.D.
Connecticut Medicine
160 St. Ronan Street
New Haven, CT 06511

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

WHO SAYS YOU CAN'T JUGGLE IT ALL ?

ProMed says you can. As a medical provider you strive to keep your patients healthy. As a businessman you aim to run an organized and efficient office. At ProMed, we can provide you with a medical office system to meet your most basic business needs — or we can provide you the advantages of a multi-user, multi-tasking operating system. And, as your practice grows, so can your ProMed office system to meet your changing needs.

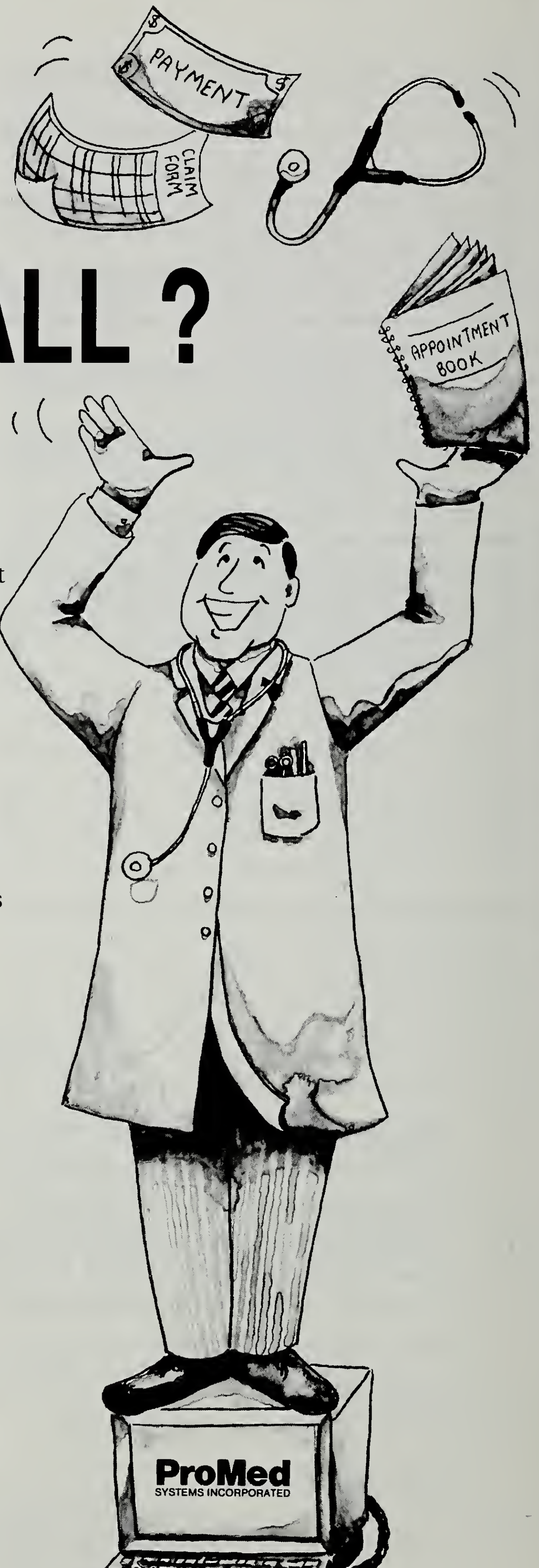
With ProMed, dedicated service and comprehensive training are only the beginning:

- ☑ Faster and more accurate electronic claims submission
- ☑ Reduced paperwork
- ☑ Detailed financial and clinical management reporting
- ☑ Complete installation and support services

As the leading provider of medical office systems in Connecticut, ProMed saves time and money for more than 1,000 of the state's medical professionals.

You take care of your patients, we'll take care of the rest. For more information, call Bob Seekamp at ProMed: 1-800-922-3385.

ProMed
SYSTEMS INCORPORATED
221 Whitney Avenue
New Haven, Connecticut 06511



Social Correlates of Malnutrition Among Filipino Street Children

ALLEN L. HIXON, M.A.

ABSTRACT—This study has sought to identify subgroups of street children at high risk for nutritional deficiency in metropolitan Manila in the Philippines. A sample of 150 street children ages six through 18 were weighed, measured, and interviewed to establish nutritional parameters and to explore the interrelationships of nutritional status with social conditions. Two variables showed the highest association with low weight for age: children using drugs and children not in school. These variables should be seen as indicative of a range of variation among Filipino street children, in whom those on drugs and not in school represent the sector at greatest risk and one to which nutritional programs should be targeted.

Background

BY the year 2000 nearly half of the world's population will live in urban areas.[1] Approximately one third of this population will be under 15 years of age. Whether in Bridgeport or Manila, urban growth and the accompanying social and environmental pressures often lead to disruption of the traditional familial and economic patterns. In the Philippines these altered patterns have forced a segment of children to leave or be pushed out of the home. Some children leave home to contribute to the economic survival of the family, others in search of a more fulfilling life, or to escape abusive family situations.

It is estimated that 50,000 to 75,000 street children live in metropolitan Manila.[2] These street children are visible in city centers begging from tourists and taking part in semilegal or illegal activities to piece together a livelihood. They are supported by their own cleverness as well as by street gangs and syndicates. A less visible group depends in part on religious and other nongovernmental residential and educational agencies. Both groups of children occasionally seek support from family or relatives, and most participate to some degree in public schooling.

A number of studies estimate that nearly half of Manila's preschool population is malnourished to the first degree. About 25% exhibit moderate malnutrition, while 4% are severely malnourished.[3] These distressing statistics arise from a historical process which began with the period of reconstruction following the devastation of World War II. Through the 1950s and 1960s the Philippines experienced a long period of industrialization and urban growth. This urbanization was concentrated in Manila and several other smaller cities.

With the Marcos era a period of accelerated growth and economic expansion was felt nationwide. However, the country's feudal roots, large regions of monocrop agriculture, and the inability to actualize a land reform program persisted. By the late 1970s this pattern of growth turned to one of stagnation. Perceived opportunities in the urban areas drew people from the depressed countryside, yet urban job creation could not keep pace with the influx of a largely untrained workforce. In the 1980s a global recession coupled with political instability, a mounting national debt, severe inflation, and the collapse of the world sugar market penalized both urban and rural Filipinos. The urbanization trend continued and by 1989, 41% of the country's population lived in urban areas.[4]

The condition of the street children in this study, as well as, among the growing numbers throughout the world, is largely a result of this global process of urbanization, a process that will undoubtedly continue.

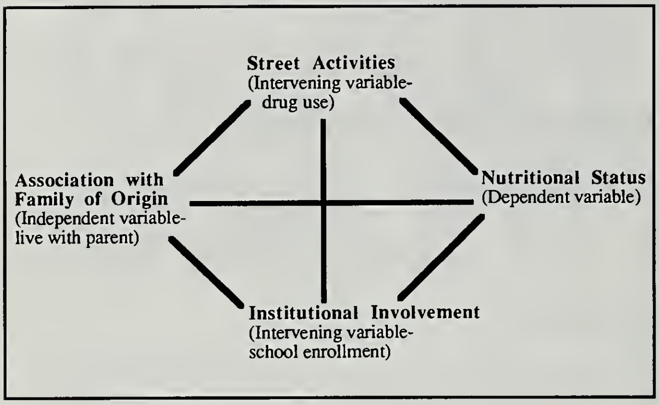
Objectives

This study examined the nutritional status of street children and correlated nutritional status with social conditions, including family of origin, institutional involvement, and street activities. The specific aim was to iden-

---

ALLEN L. HIXON, M.A., University of Connecticut School of Medicine.

tify high-risk subgroups that could be targetted with nutritional interventions.



The hypotheses derived from this model are:

- Street children as a group are malnourished in relation both to Filipino and international standards.
- Higher levels of involvement in societal institutions indicate better nutritional status.
- Closer ties to family of origin indicate better nutritional status.
- Less involvement in negative street activites (drugs, gangs, crime) the better the nutritional status.

Thus the factors of level of involvement with societal institutions, degree of association with family of origin, and aspects of street life affect nutritional status.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Methods and Research Design

One neighborhood estimated to have 500 street children was studied. A series of interviews with service providers, street children, and families was conducted. Secondary census data and growth charts were gathered from the Food and Nutrition Research Institute. Based on this information, a 34-question, native language interview was written, translated, and pretested. The four major agencies working with street children in this community provided the sampling base. Social workers from four agencies provided introductions to the various gangs and syndicates of children.

A stratified, purposive sample was drawn including 150 males and females, ages six through 18. Street-based, center-based, and community-based (squatter settlement) children were included. Anthropometric and interview data were obtained from approximately 25% of the estimated population of street children in the community being studied. Staff from the Department of Health cooperated in conducting the interviews. Height was measured with a standard tape measure and weight was determined by a callibrated bathroom scale.

The weight, height, and age data were converted to an international weight/age and height/age *z* score by the Centers for Disease Control—National Center for Health Statistics (CDC-NCHS) standards.[3] Low mean weight/age *z* score suggests that a population exhibits acute undernutrition while low mean height/age *z* score suggests chronic undernutrition.

Squatter settlement.

Results

Males and females of all ages were underweight and underheight by both international and Filipino standards. (Mean weight/age *z* score = -1.50, mean height/age *z* score = -2.02, NCHS). This is equivalent to the lowest 8% for weight and the lowest 2.5% for height compared to the the international standard. Gender showed no correlation to nutritional status. The weight/age *z* score was found to be a more useful measure for defining high-risk subgroups in this population and was utilized in subsequent correlations.

Independent Variables

Several measures of each of the independent factors were assessed in terms of their relationship to the dependent factor of nutritional status. For the factor of institutional involvement, one variable which emerged as associated with nutritional status was current school attendance which correlated significantly with increased weight/age *z* score. (*P*=0.002, n=112, *t*-test).

For the factor of street life-style, drug use was a key variable which emerged. Having tried dangerous drugs (downers, glue) correlated significantly with a decreased weight/age *z* score. (*P*=0.0004, n=104, Mann-Whitney *U*).

Young girl living on sidewalk.

Street boy collecting plastic and metal scraps to recycle.

The variables of school enrollment and drug use were highly interrelated (those enrolled in school were less likely to use drugs, χ^2, $P=.001$). The two variables combined showed a stronger relationship to nutritional status (school enrollment $P=.017$, drugs $P=.007$, combined $P<.001$, ANOVA). Those who study and do not use drugs show the highest mean weight. Those who are not enrolled in school and who use drugs show the lowest mean weight, while those who exhibit one behavior (no school or drugs) fall in between.

While relation to family of origin was not directly linked to weight/age *z* score, close association with family of origin correlated with school enrollment (χ^2, $P=.036$) and with less drug use (χ^2, $P=.00002$). Although by itself not predictive of nutritional status, family appears as a key variable affecting street children's life-styles and therefore working indirectly through these factors.

Analysis and Conclusions

These data suggest that both chronic and acute undernutrition exist in this population. In many cases these conditions are superimposed presenting as a stunted underweight child. Higher-risk subgroups exist, and there is a considerable range of variation across this population both in terms of anthropometric measures and life-style. While these findings are not surprising, what is most important is how insight into behavioral patterns can be used to identify high-risk subgroups of children and to structure appropriate nutritional interventions.

One subgroup, those who are not in school and have used dangerous drugs, are at risk for low weight-for-age. School enrollment for these children appears to represent a package of positive behaviors. School involvement is likely to provide a daily structure, a meal plan, and a disciplined environment that these childen are missing.

Approximately half of the respondents reported using some combination of speed, downers, solvent, and "rugby," an inexpensive glue. Glue sniffing was the most common and accessible addictive behavior among this subgroup. Patterns of drug use may identify a hard-core street child that is more likely to be malnourished. Glue sniffing has been reported to curb the appetite and therefore may be more commonly used by the most hungry children.

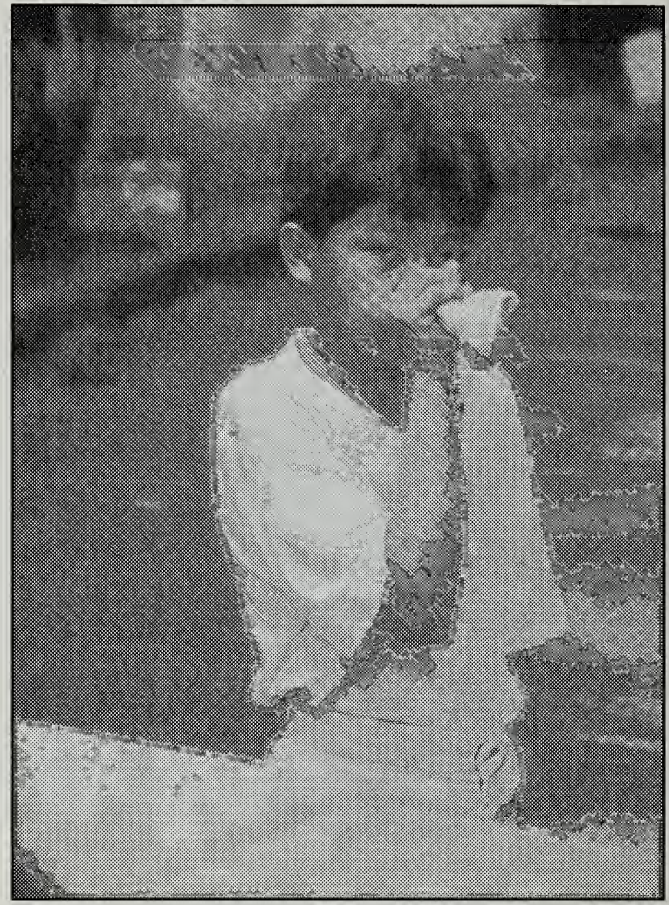
As noted above, relation to family of origin does not correlate directly with nutritional status. Families of street children are generally very poor. Children may exhibit low weight and height prior to leaving the home. In addition, the relation of children to their families made up a continuum. Some children lived part time at home or split time between parents, relatives, and friends. Others lived on the street or in an institution for a short period before returning home. Similarly, regarding school and street activities, there was tremendous variability and movement between activities. While begging, vending, scavenging, and prostitution were all common, on any given day, a child might wake up and sell newspapers, spend half a day in school, scavenge and recycle in the afternoon, and beg in the tourist district at night. It is necessary to understand this level of complexity and the fluidity of each parameter that was studied.

Although few longitudinal studies are available, clearly the health of street children is at significant risk. Feedback on children identified by this study have described serious medical problems including tuberculosis of the hips, hepatitis, and cardiac failure. UNICEF and many national organizations are concerned about the quality of life of street children. Programs have been implemented to strengthen family structures, support children in school, and to provide street drop-in centers, meals, and a place for children to sleep.

This study suggests that the child at greatest nutritional risk is the hardcore street child who is not in school and uses drugs. These children are tough, mobile, and least likely voluntarily to enter a structured program. Well-intentioned programs that require drug-free behavior are likely to miss the children at greatest risk. Similarly, strengthening family ties, while an appealing approach, is unlikely to show direct nutritional improvement at the level of the individual child.

Further studies looking at the variables of mobility, school enrollment, and drug use should be undertaken in other Manila neighborhoods. Programs that are currently serving street children should systematically assess barriers to participation by high-risk children in their communities. Pilot projects to train current street educators and social workers in substance abuse counseling should be put in place. Finally, a detailed study should be made of the territories, activities, and membership of the various gangs and syndicates of children in Manila. Understanding this functional level of organization for many street children may provide a route to reach children at significant nutritional risk. Only armed with a realistic understanding of the behaviors of the various subgroups of street children can unique and effective nutritional outreach programs be targetted.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE



Sniffing "rugby" glue.

Acknowledgements

Special thanks to UNICEF, The Philippines Country Office for their support and assistance, the University of Connecticut Health Center Research Advisory Committee for the provision of a summer research stipend, the International Education Committee of the University of Connecticut Health Center and the Connecticut Association of Philippine Physicians for their provision of travel funds, and to my faculty preceptor, Stephen L. Schensul, Ph.D. of the Department of Community Medicine, University of Connecticut School of Medicine.

REFERENCES

1. Gugler J, ed: *The Urbanization of the Third World.* New York, Oxford University Press; 1988:1.
2. *The Situation of Street Children in Ten Cities*, UNICEF, Manila, Philippines, 1988.
3. National Center for Health Statistics, Atlanta, Ga: Centers for Disease Control.
4. Black M, *Philippines: Children of the Runaway Cities*, UNICEF, Florence, Italy, 1991.

Important Advances in Clinical Medicine

Neurosurgery

The Council on Scientific Affairs of the California Medical Association presents the following inventory of items of progress in neurosurgery. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome, and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, researchers, and scholars to stay abreast of these items of progress in neurosurgery that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Neurosurgery of the California Medical Association, and the summaries were prepared under its direction.

Carotid Endarterectomy

THE first carotid artery endarterectomy was done in 1954 on a patient with ipsilateral cerebral transient ischemic attacks. By 1971, 15,000 endarterectomies had been done in the United States, and this number rose to 107,000 in 1985. Although many vascular surgeons and neurosurgeons were convinced of the benefits of this operation, others were skeptical. The 1991 publication of three prospective, randomized, well-designed, and well-executed trials—the North American Symptomatic Endarterectomy Trial (NASCET), the European Carotid Surgery Trial (ECST), and the Veterans Affairs (VA) Symptomatic Carotid Stenosis Trial No. 309—showed unequivocally that surgical intervention is highly beneficial for symptomatic patients with tight carotid stenosis of 70% or more. These remarkable studies have elicited a wealth of new data concerning the natural history of symptomatic carotid artery disease and its treatment with carotid endarterectomy.

The NASCET study showed that carotid endarterectomy reduces the risk of stroke ipsilateral to the stenosis at two years from 26% to 9%, a relative risk reduction of 65% and an absolute risk reduction of 17% compared with the aspirin-only treatment group. The VA study showed an absolute risk reduction of 11.7% in patients with carotid stenosis of greater than 50% and 17.7% in patients with stenosis of 70% or more. The ECST study showed a similar benefit for surgical treatment (85% relative risk reduction). The benefit of surgical treatment in reducing stroke appeared early, within three months, and persisted for at least three years. For major or fatal ipsilateral stroke, surgery provided a relative risk reduction of 81% (13% for medical treatment compared with 2.5% for surgical). Furthermore, the absolute risk reduction for ipsilateral stroke offered by surgical therapy varied directly with the degree of stenosis (26% for 90% to 99%, 18% for 80% to 89%, and 12% for 70% to 79%).

The general conclusion from these studies is that patients with symptomatic carotid stenosis of 70% or greater should be operated on. It is important, however, to consider the inclusion criteria for these trials: transient ischemic attack, minor stroke or nondisabling major stroke of recent onset (less than four months in NASCET, less than six months in ECST), and a life expectancy of five years. The NASCET trial excluded patients with severe intracranial atherosclerotic stenoses, those with major organ failure (kidneys, liver, or lungs), a possible cardioembolic source, or previous ipsilateral carotid endarterectomy.

Because data from several studies have suggested that duplex ultrasound scanning does not always correlate with angiography, a decision regarding surgical intervention should be based on angiography, including the

---

Reprinted from *The Western Journal of Medicine*, January 1993.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

intracranial circulation. The size of internal carotid artery stenosis should be calculated from the luminal diameter at the site of the greatest degree of stenosis compared with the artery diameter beyond the plaque and carotid bulb (NASCET criteria) or with respect to the presumed diameter of the carotid bulb (ECST criteria). The sensitivity and specificity of newer noninvasive imaging techniques—magnetic resonance angiography and rapid spiral computed tomography for carotid artery disease—remain to be determined. If these noninvasive methods prove to be reliable measures of carotid stenosis, they may become extremely useful in the future.

The perioperative major stroke and death rate was only 2.1% in the NASCET study. This reflects the skills that have evolved in surgeons with experience in working with modern equipment and sophisticated anesthesia. Technical improvements, such as intraoperative cerebral monitoring—electroencephalography, somatosensory evoked potentials, cerebral blood flow measurements, and internal carotid stump pressures—as well as the use of intraoperative shunts, magnification, and cerebral protective agents, such as barbiturates and mannitol, have been helpful to various surgical teams, although their use was not shown to decrease or increase the risk of neurologic complications. These adjunctive techniques should be used according to the experience of the surgeon and the circumstances dictated by each case. In patients in whom there is concern about collateral flow during internal carotid clamping (for instance, contralateral carotid occlusion and small posterior communicating arteries, or a change in the intraoperative cerebral monitoring variables), the use of an internal shunt or induced hypertension would be warranted. Obviously hypotension should be avoided during temporary internal carotid artery occlusion.

Whether patients with symptomatic carotid stenosis of 30% to 60% will benefit from endarterectomy is not yet known as the prospective studies have not been completed. At present, such patients should be randomly assigned to one of these trials or a decision regarding surgical therapy made based on the ischemic event, the medical condition of the patient, and the angiographic appearance of the lesion. For instance, the presence of a 60% symptomatic stenosis with an ulcer seen on angiography might weigh more heavily in favor of surgical treatment since the NASCET study showed that ulcers in association with stenosis increased the risk of ipsilateral stroke and that surgery was highly beneficial for such patients. There is no role for surgical intervention in patients with symptomatic stenosis less than 30% because the ECST study showed that endarterectomy actually worsened the results at three years—3.3% ipsilateral stroke in the surgical treatment group compared with 1.3% without an operation.

Two trials—VA Asymptomatic Trial No. 167 and the Asymptomatic Carotid Atherosclerosis Study—are attempting to address another unresolved issue, asymptomatic carotid stenosis. Although it is tempting to generalize the results of the studies on symptomatic patients with high-grade stenosis to asymptomatic patients with similar lesions, this is not yet warranted. The one published study of asymptomatic patients with carotid stenosis of 50% to 90% (Carotid Artery Surgery Asymptomatic Stenosis—Operation Versus Aspirin) showed no benefit of surgical therapy compared with aspirin therapy in these patients. Serious methodologic flaws in the study suggest that the issue remains in doubt, however. Until further data become available, the treatment of patients with asymptomatic carotid stenosis must be carefully tailored to each patient, with a tendency toward conservative management unless the stenosis is severe.

Gary K. Steinberg, M.D., Ph.D.
Stanford, California

John A. Anson, M.D.
Albuquerque, New Mexico

REFERENCES

Bousser MG: Benefits from carotid endarterectomy? Yes, but .... *Cerebrovasc Dis* 1992; 2:122-6.

CASANOVA Study Group: Carotid surgery versus medical therapy in asymptomatic carotid stenosis. *Stroke* 1991; 22:1229-35.

Ferguson GG: Extracranial carotid artery surgery. *Clin Neurosurg* 1992; 29:543-79.

Mayberg MR, Wilson SE, Yatsu F, et al: Carotid endarterectomy and prevention of cerebral ischemia in symptomatic carotid stenosis—Veterans Affairs Cooperative Studies Program 309 Trialist Group. *JAMA* 1991; 266:3289-94.

North American Symptomatic Carotid Endarterectomy Trial Collaborators: Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 1991; 325:445-53.

The Limits of Treatment of Malignant Gliomas

THE diagnosis of malignant glioma—anaplastic astrocytoma and glioblastoma multiforme—has traditionally been associated with a sense of hopelessness for health care professionals as well as patients. Physicians have been taught that the prognosis is uniformly poor regardless of the treatment. This has led to a conservative approach for many patients with malignant glioma, with survival ranging from 12 to 38 weeks.

Recent experimental treatments have failed to extend survival for patients with malignant gliomas. Despite a lack of effective new treatment options, subgroups of patients are "long-term" survivors with standard multimodality therapy. The current clinical challenge is twofold: the selection of patients who are good candidates for multimodality therapy, and the maintenance of quality, not quantity, of life for these patients.

Many factors influence the prognosis of malignant gliomas. Age is probably one of the strongest predictors of survival, as patients aged 18 to 44 years have a median survival of 107 weeks, whereas patients older than 65 have a median survival of 23 weeks. Neurologic function at the time of diagnosis, a history of a previous low-grade tumor, and the histologic composition of the tumor also affect the prognosis substantially. The treatments that influence survival are radiation therapy, surgical therapy, and chemotherapy.

Patients who undergo a complete resection of the tumor, as seen on computed tomography (CT) or magnetic resonance imaging (MRI) scans, have a median survival of 70 weeks, in contrast to a median survival of 19 weeks in patients who undergo only biopsy of the tumor. Magnetic resonance imaging and CT scans do not identify the outermost border of malignant brain tumors; areas that appear "normal" on these imaging studies have been shown to contain tumor cells. Surgical therapy is therefore not curative for high-grade gliomas. An improved ability to identify tumors intraoperatively and map eloquent cortical functions, such as language and motor areas, allows safe surgical resection and cytoreduction in an increasing number of previously "inoperable" patients. Despite no evidence of tumor on postoperative imaging studies, almost all patients have recurrences; 80% of these recurrences will be within 2 cm of the resection margin.

Radiation therapy is still the most effective treatment for these tumors. Regional irradiation in the range of 60 Gy (3 cm beyond the tumor border seen on CT or MRI) is given at most institutions. To preempt recurrences at surgical margins, some centers have recently begun protocols using radioactive seeds (brachytherapy) or stereotactic radiosurgery to boost the tumor margins with single doses as high as 20 to 30 Gy. Most studies using chemotherapy have shown only minimal benefit. Individual patients can respond dramatically to chemotherapy, however, with imaging resolution of the tumor and improved neurologic function. A third of patients will have some response to chemotherapy. In young patients, particularly those with anaplastic astrocytoma, adjuvant chemotherapy with procarbazine hydrochloride, lomustine, and vincristine sulfate has proved beneficial and is extremely well tolerated.

In patients younger than 65 who still enjoy good function, maximum surgical resection, followed by radiation therapy, with a boost to the resection margin, and a trial of adjuvant chemotherapy is currently the most likely approach to maintain quality of life and extend survival. It is essential to observe this group of patients closely with imaging studies repeated every two to three months. Single photon-emission CT scans using thallous chloride T1 201 are extremely effective in detecting tumor recurrences and in distinguishing recurrence from radiation necrosis. To maintain function and quality of life, it is important to intervene early when tumor recurrence is the likely diagnosis. A second surgical resection or stereotactic radiosurgery can sometimes be effective. Stopping or changing chemotherapy should also be considered. Patients for whom these treatments fail should be considered for experimental approaches, again with the caveat that quality, not quantity, of life should be the goal of treatment.

Strategic planning of an aggressive multimodality treatment approach is best achieved through the collaboration of a "brain tumor team" that includes neurosurgeons, radiation oncologists, medical oncologists, and other health care professionals. This ensures that the patient receives the maximum benefit available from the multidisciplinary treatment of malignant gliomas.

Keith L. Black, M.D.
Joseph R. Ciacci, M.D.
Los Angeles, California

REFERENCES

Black KL, Hawkins RA, Kim KT, et al: Use of thallium-201 SPECT to quantitate malignancy grade of gliomas. *J Neurosurg* 1989; 71: 342-6.

Brada *M:* Radiosurgery for brain tumors. *Eur J Cancer* 1991; 27:1545-8.

Levin VA, Silver P, Hannigan J, et al: Superiority of post-radiotherapy adjuvant chemotherapy with CCNU procarbazine, and vincristine (PCV) over BCNU for anaplastic gliomas—NCOG 6G61 final report. *Int J Radiat Oncol Biol Phys* 1990; 18:321-4.

Indications and Limitations of Stereotactic Radiosurgery

STEREOTACTIC radiosurgery is an important new treatment option for many patients with brain lesions. Although radiosurgical procedures have been used in a limited way for more than 20 years, the development of computerized imaging—computed tomography and magnetic resonance imaging—has created many new applications. By coupling stereotactic localization techniques with a sharply collimated beam, specialized instrumentation makes it possible to target precisely an ablative dose of ionizing radiation to a well-circumscribed brain lesion. With an accuracy approaching 1 mm, a large "necrosing" dose of radiation (10 to 50 Gy) is delivered at one time. Radiosurgical treatment often supplants a craniotomy and is typically done on an outpatient basis. The three basic types of radiosurgical technique are distinguished by radiation source: heavy charged particle; gamma knife, which uses 201 cobalt 60 sources; and linear accelerator-based systems. Radiophysical and clinical studies suggest that under most circumstances the three techniques are comparable.

Radiosurgery has proved to be a valuable tool for treating many benign lesions. For arteriovenous malformations, a single treatment produces intimal hypertrophy and gradual angiographic obliteration over one to two years in 85% to 90% of patients. This result affords permanent protection against rebleeding. During the latent period, however, the risk of rehemorrhage is unaffected. The major complication of radiosurgical treatment of arteriovenous malformations is delayed radionecrosis. The reported risk of this problem ranges from 3% to 9% and is proportional to the volume being irradiated. Smaller but equally effective doses of radiation are currently being used, and the incidence of complications is decreasing. Given the good results that can be achieved with standard neurosurgical resection, radiosurgery is the treatment of choice for small, poorly accessible arteriovenous malformations or patients in poor medical condition.

Radiosurgical ablation is also being used increasingly to treat benign tumors such as acoustic neurinoma and unresectable or recurrent skull base meningioma. By delivering a single large dose of radiation, stereotactic radiosurgery prevents tumor growth in greater than 95% of patients with acoustic neurinomas, and, in 40% of patients, tumor shrinkage occurs. Radiosurgery is associated with transient facial weakness or facial sensory disturbance in 30% of patients. Nevertheless, hearing can sometimes be preserved, especially with smaller lesions. Although radiosurgery prevents symptomatic and radiographic progression in nearly all cases of meningioma, the follow-up of such patients is only a few years. The proximity of the radiation-sensitive optic nerves or chiasm limits the aggressiveness of the radiosurgical treatment of recurrent or unresectable pituitary tumors. New radiosurgical instrumentation and computerized imaging techniques have renewed interest in treating these lesions by these methods. Although radiosurgery has also been used to treat several other types of benign tumors such as hemangioblastoma, craniopharyngioma, and neoplasms of the pineal gland, the benefits of therapy are largely anecdotal. Overall, radiosurgery can be helpful for managing many challenging small benign brain tumors.

Although radiosurgery has been used for only a brief time to treat malignant brain tumors, results have been encouraging. In preliminary studies, a stereotactic "boost" has been used to treat intrinsic malignant gliomas. Furthermore, several investigators have reported impressive local tumor control and minimal morbidity using radiosurgery to treat metastatic brain lesions. Given the relative ease of radiosurgical ablation, this treatment may supplant standard techniques of removing tumors. Clinical trials are in progress to investigate this hypothesis. Ultimately the most widespread use of radiosurgery may be for the management of malignant neoplasms.

John R. Adler, Jr, M.D.
Stanford, California

REFERENCES

Adler JR, Cox RS, Kaplan I, Martin DP: Stereotactic radiosurgical treatment of brain metastases. *J Neurosurg* 1992; 76:444-9.

Lundsford LD, Flickinger J, Coffey RJ: Stereotactic gamma knife radiosurgery— Initial North American experience in 207 patients. *Arch Neurol* 1990; 47:169-75.

Steinberg GK, Fabrikant JI, Marks MP, et al: Stereotactic heavy-charged-particle Bragg-peak radiosurgery for intracranial arteriovenous malformations. *N Engl J Med* 1990; 323:96-101.

Steiner L, Lindquist C, Adler JR, et al: Clinical outcome of radiosurgery for cerebral arteriovenous malformations. *J Neurosurg* 1992; 77:1-8.

Corpus Callosotomy for Intractable Epilepsy in Children

THE surgical treatment of medically intractable seizures is now a well-established and expanding aspect of the management of adults with epilepsy. In many cases, the focal epileptogenic area of the brain is first localized by scalp or intracranial electroencephalographic (EEG) recordings and then is excised by craniotomy. The most common operation for seizure in adults is the temporal lobectomy, since partial seizures originating there are relatively common and are frequently difficult to control with anticonvulsants.

Surgical therapy for medically intractable seizures is now being done on patients in the pediatric age range. This is due to several factors. There have been substantial improvements in seizure monitoring and implantable electrode technology. There is mounting evidence to suggest that surgical intervention must occur relatively early in life, before the educational and social deprivation that typically cripples persons with long-standing uncontrolled seizures. In addition to the relatively rare partial epilepsies, a common epileptic syndrome in children is symptomatic generalized epilepsy. This syndrome may arise as a result of widespread, even bilateral, regions of epileptogenesis and is characterized by a bilateral onset of seizures as recorded by EEG. These attacks occur as atonic "drop attacks," tonicseizures, and tonic-clonic seizures as well as myoclonic, absence, and akinetic

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

seizures. These epilepsy syndromes do not lend themselves to traditional surgical resection because there is not a restricted epileptogenic focus.

In 1940 the corpus callosotomy was introduced as an operation designed to treat seizures. The theory was that the bihemispheric spread or "synchrony" of the seizure discharge was, in part, dependent on the corpus callosum and the anterior commissure that bound the two hemispheres together. The operation did not receive much attention and was criticized for several reasons: acute severance of the entire corpus callosum produced a profound neuropsychological deficit, a syndrome of "disconnection" consisting of unilateral tactile anomia, left hemialexia, mutism, and unilateral apraxia; and complications of the surgical procedure were high (aseptic meningitis and hydrocephalus were seen after the operation in more than 33% of patients).

In the past decade, most of these problems have been overcome. The neuropsychological deficit is reduced by limiting the sectioning of the corpus callosum to the anterior two thirds. The complications have been mitigated by the use of microsurgical techniques and steroids. The procedure has now become an important tool in the surgical management of generalized seizure disorders, particularly in children. A substantial reduction in seizure frequency has been reported in 78% to 84% of patients undergoing corpus callosotomy for the treatment of generalized and mixed seizures. Although generalized seizures do respond to callosal section, as many as a third of patients with concomitant simple and partial seizures may show an increase in these seizures. Morbidity and mortality have been low. In patients who do not respond to the initial sectioning, there is evidence both for and against proceeding with complete section of the corpus callosum (posterior third).

Kim J. Burchiel, M.D.
Portland, Oregon

REFERENCES

Fuiks KS, Wyler AR. Hermann BP, Somes G: Seizure outcome from anterior and complete corpus callosotomy. *J Neurosurg* 1991; 74: 573-8.

Nordgren RE, Reeves AG, Viguera AC, Roberts DW: Corpus callosotomy for intractable seizures in the pediatric age group. *Arch Neurol* 1991; 48:364-72.

Spinal Cord Injuries in Children Without Radiographic Abnormalities

CHILDREN are susceptible to spinal cord injuries without concurrent radiographic evidence of vertebral column disease. In contrast, adult spinal cord injuries are typically accompanied by radiographically detectable fractures or ligamentous spinal injuries. Spinal cord injury without radiographic abnormalities is defined as objective signs and symptoms of myelopathy with normal plain radiographs and computed tomographic (CT) studies. Injuries range from mild transient neurologic symptoms—such as spinal cord concussions—to permanent complete paralysis.

Spinal cord injury without radiographic abnormalities accounts for 30% of spinal injuries in children eight years of age and younger and for 10% in children between nine and 16 years old. Young children usually sustain severe neurologic injury; 75% of the injuries in this age group are associated with a complete loss of spinal cord function. Older children (nine to 16 years) usually sustain less severe, typically incomplete neurologic injuries. The cervical or thoracic spinal cord is involved with equal frequency; lumbar injuries are rare.

Although most injuries occur immediately with trauma, children can have a delayed onset of neurologic deficits. Brief, transient motor and sensory symptoms associated with a mild injury are often initially reported. An asymptomatic period can intervene, and after several hours or days, neurologic deficits can develop. Patients who have a delayed occurrence of neurologic deficits tend to have severe or complete neurologic spinal cord syndromes, which may represent spinal cord infarction.

Spinal cord injuries in children should be investigated with thorough radiographic and imaging studies to exclude treatable injuries. The osseous structures should be evaluated by plain radiography and CT. Thin-section CT studies are directed two vertebral levels above and below the clinical level of neurologic injury. Magnetic resonance imaging or myelography is indicated to evaluate for treatable compressive lesions, such as disc herniations or hematoma. Magnetic resonance imaging is preferred because it is noninvasive and precisely delineates spinal cord and soft tissue anatomy and abnormalities.

The pathogenesis of spinal cord injuries without radiographic abnormalities arises from the elasticity and hypermobility of immature spines. The vertebral column in young children is flexible and composed of structurally weak, architecturally immature components. Mechanisms of injury include direct spinal cord traction or compression, an indirect transmission of kinetic energy to the spinal cord, or vascular or ischemic mechanisms.

Although rare, treatable causes such as occult fractures, spinal epidural hematomas, disc herniations, occult subluxations, or spinal instability must be considered.

The initial treatment of a patient with documented myelopathy includes maintaining continuous external spinal immobilization and assessing for treatable lesions. A Philadelphia collar or a more rigid brace is used for cervical injuries until neurologic deficits have resolved. Subsequent flexion-extension views of the cervical spine are necessary to rule out latent ligamentous instability. Activity is strictly limited for at least three months because of the risk of recurrent injury. Patients with spinal cord injuries at the thoracic or lumbar level without radiographic abnormalities are initially treated with bed rest and gradual mobilization. Dynamic radiographs are obtained to rule out occult ligamentous instability or late spinal deformity.

A third of children sustain recurrent injuries. Recurrent spinal cord injury without radiographic abnormalities is a serious problem that usually occurs in children with mild initial deficits, the severity of which has been underestimated. These children disregard their immobilization devices and resume normal activities too early. Recurrent injuries tend to result in profound deficits with permanent sequelae.

It has been suggested that children are capable of recovering more function after spinal cord injury than adults with comparable injuries. This notion is based on the unsubstantiated hypothesis that children possess greater plasticity of their central nervous system than adults. The prognosis for these injuries, like the prognosis for all other spinal cord injuries, is related to the severity of the initial injury. Patients with a complete loss of sensory and motor function have a poor prognosis. Children with incomplete neurologic injuries have a better long-term prognosis that depends directly on the severity of the initial motor or sensory deficits. The objectives of therapy are to provide the maximum potential for neurologic recovery and to prevent recurrent injury. Steroid therapy has not been studied specifically in these patients. Based on the results of treating other patients with myelopathy with methylprednisolone, however, we strongly recommend high-dose intravenous methylprednisolone sodium succinate therapy for patients with spinal cord injury regardless of the cause or radiographic findings. Methylprednisolone should be administered intravenously as a bolus of 30 mg per kg of body weight followed by an infusion at 5.4 mg per kg per hour for 23 hours.

Curtis A. Dickman, M.D.
Joseph M. Zabramski, M.D.
Harold L. Rekate, M.D.
Volker K. H. Sonntag, M.D.
Phoenix, Arizona

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

REFERENCES

Bracken MB, Shepard MJ, Collins WF, et al: A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. *N Engl J Med* 1990; 322:1405-11.

Dickman CA, Zabramski JM, Hadley MN, Rekate HL, Sonntag VKH: Pediatric spinal cord injury without radiographic abnormalities: Report of 26 cases and review of the literature. *J Spinal Disord* 1991; 4:296-305.

Pollack IF, Pang D, Sclabassi R: Recurrent spinal cord injury without radiographic abnormalities in children. *J Neurosurg* 1988; 69:177-82

Zwimpfer TJ, Bernstein M: Spinal cord concussion. *J Neurosurg* 1990; 72:894-900.

Management of Growth Hormone-Secreting Pituitary Adenomas—Early Diagnosis Leads to Successful Treatment

ACROMEGALY is the least subtle of the hypersecretion syndromes from pituitary adenomas, although it is frequently missed in the early stages of the disease. Not only is the disease disfiguring, it is also disabling and has substantial associated morbidity and mortality. Consequently, the successful management of patients with this disease is dependent on the prompt and proper integration of surgical and medical therapies.

The clinical features of growth hormone (GH) hypersecretion are well known to most clinicians and include coarsening of facial features, enlargement of the hands and feet, and increased water retention within soft tissues. The diagnosis of acromegaly is made by showing elevated basal serum GH and somatomedin C levels along with radiographic confirmation of the presence of a tumor in the sella turcica on magnetic resonance imaging. Occasionally hormonal values will be normal in patients with symptoms suggestive of acromegaly. In these cases, the diagnosis can be confirmed by demonstrating nonsuppression of the serum GH levels during an oral glucose tolerance test. A normal response is a fall in the GH level to below 2 ng per mL after 75 to 100 grams of oral glucose. Similar criteria are used to assess the adequacy of treatment after surgical intervention.

Surgical resection remains the primary mode of treatment of patients with GH-secreting pituitary adenomas. In most cases, a transsphenoidal operation can achieve complete extirpation of the tumor with preservation of normal pituitary function. Only rarely, if the tumor is extraordinarily large, will a craniotomy be required. Resection is contraindicated in severely debilitated patients or when pituitary hyperplasia from a concomitant hypothalamic lesion is suspected. Basal GH levels are expected to return to normal in 60% to 90% of patients after surgical treatment, most frequently when the tumor is a microadenoma (less than 1 cm in diameter) and when the preoperative hormone level is less than 40 ng per mL. In this group, acromegalic symptoms will be relieved and normal pituitary function preserved in greater than 90% of patients.

The medical management of acromegaly is based on the physiologic regulation of GH secretion. An analogue of somatostatin, the natural inhibitor of GH release, has recently been developed that has been shown to cause short- and long-term reduction of GH levels when given in 100-μg subcutaneous injections two or three times a day. In about 50% of people with acromegaly, GH and somatomedin C levels return to normal following octreotide acetate treatment; the associated symptoms and signs are alleviated in 80% of patients. The principal side effect of the medication has been gastrointestinal irritability, and its use has been associated with an increased incidence of gallstones. Although tumor size does decrease in response to octreotide therapy, the effect is not as rapid nor as pronounced as the response of prolactin-secreting macroadenomas to the administration of bromocriptine. The present indications for octreotide therapy include tumors that are not cured by surgical therapy and patients who are not considered surgical candidates. Octreotide may also play a role as a preoperative adjunct to aid the surgical removal of tumor in the future.

Radiation therapy can be considered in patients who do not respond to surgical and medical therapy. Conventional techniques using a total dose of 45 to 50 Gy have been recommended. A hormonal response, however, may take as long as several years and frequently is associated with panhypopituitarism. Stereotactic radiosurgery using the linear accelerator or gamma knife has recently been used to precisely irradiate selected intracranial neoplasms. Several patients with pituitary tumors have been treated, but long-term results are not available at the present time.

The sequelae of acromegaly, including disfigurement, hypertension, cardiomyopathy, and death, underscore the need for the urgent treatment of these patients. In many cases the physical signs and symptoms are reversible. Improved surgical techniques and new medical strategies may allow more effective management in the future.

Wesley A. King, M.D.
Los Angeles, California

REFERENCES

Barkan AL, Kelch RP, Hopwood NJ, Beitins IZ: Treatment of acromegaly with the long-acting somatostatin analog SMS 201-995. *J Clin Endocrinol Metab* 1988; 66:16-23.

Lamberts SWJ, Uitterlinden P, del Pozo E: SMS 201-995 induces a continuous decline in circulating growth hormone and somatomedin-C levels during therapy of acromegalic patients for over two years. *J Clin Endocrinol Metab* 1987; 65:703-10.

Ross DA, Wilson CB: Results of transsphenoidal microsurgery for growth hormone-secreting pituitary adenoma in a series of 214 patients. *J Neurosurg* 1988; 68:854-67.

Interventional Neuroradiology—A New Approach to Difficult Vascular Lesions of the Brain

INTERVENTIONAL neuroradiology has assumed an important role in the treatment of intracranial cerebrovascular lesions, particularly in high-risk patients in whom a poor surgical result would be expected. Such lesions as intracranial aneurysms, vascular malformations, vasospasm following subarachnoid hemorrhage, and thromboembolic stroke are amenable to treatment by the endovascular approach. Either as the sole therapy or in conjunction with a surgical procedure, interventional radiology is proving to be a valuable treatment alternative for difficult vascular lesions of the central nervous system.

Highly specific treatment can be administered, including but not limited to the instillation of pharmacologic agents, the injection of particulate embolic substances or glue embolization, the release of detachable microcoils, the inflation of detachable and nondetachable balloons, or a combination of these agents. This less-invasive therapy often allows patients to be discharged from hospital within three days. Some patients require several sessions of therapy, which may be spread out over a period of weeks. Other patients need only a single session. Patients undergoing endovascular therapy are usually selected because they are poor surgical risks or are those in whom conventional neurosurgical techniques are likely to be unsuccessful. As endovascular techniques improve with evolving technology and experience, endovascular therapy will likely become the prime mode of therapy for certain indications.

Intracranial aneurysms can be treated by endovascular occlusion of the aneurysm itself or its feeding artery. This offers the benefits of continuous monitoring of the patient's neurologic status during the procedure, the avoidance of general anesthesia and brain retraction, and immediate high-quality angiography for verification of the procedure's success. For these reasons, several centers have been actively pursuing coil embolization of aneurysms. Small microcoils are pushed through a microcatheter into the aneurysm itself until the aneurysm sac is virtually filled. This may require the placement of multiple coils of varying sizes and configurations. The coils are detached either mechanically or electrolytically. Pitfalls include incomplete thrombosis of the aneurysm, difficulties in access, unintentional aneurysmal rupture or vascular damage, thrombosis of normal adjacent vessels, and ischemic complications. Long-term follow-up for these patients is required. Further research is ongoing, but it is clear that this technique holds great promise.

Intracranial arteriovenous malformations are well suited to endovascular techniques. Surgical access may be difficult or impossible, and some lesions are too large for effective focused radiotherapy. To select patients suitable for treatment, we analyze angiographic risk factors predisposing to bleeding and "steal" symptoms. Endovascular embolization is especially useful as an adjunct to conventional neurosurgical and radiosurgical procedures. Advantages include continuous neurologic monitoring of awake patients, the ability to test vascular territories using intraarterial administration of amobarbital, and the staged obliteration of the larger malformations to minimize altered hemodynamics. Microcatheters are maneuvered into the feeding arterial pedicles of the arteriovenous malformations, and a rapidly polymerizing cyanoacrylate liquid adhesive is administered to thrombose the arteriovenous malformations. Many pedicles can be devascularized in one session, but the larger malformations require several sessions. A few intracerebral arteriovenous malformations may be cured by this technique, and in most lesions the size of the lesion and blood flow are substantially reduced. Limitations of the procedure are related to vascular access problems, the location of the feeding vessels in critical contiguous areas, and the risks of induced hemorrhage or infarction. In more than 100 such procedures, results have been encouraging, with longterm follow-up continuing.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

Symptomatic vasospasm after subarachnoid hemorrhage is still a challenge in neurosurgical management. Endovascular treatments include mechanical dilatation of the vasospastic areas using balloon angioplasty. This technique can be used to reach lesions in large arteries of the circle of Willis. Associated problems include possible aneurysmal rupture if the aneurysm has not already been treated, worsening ischemic changes leading to infarction, and difficulty of access to the narrowest distal arterial branches and perforators, which may be responsible for the ischemic symptoms. The endovascular treatment of vasospasm using pharmacologic agents such as intra-arterial papaverine hydrochloride and other agents may also prove useful. The longterm effects of intracranial angioplasty and pharmacologic treatment of vasospasm are not known and remain a worthwhile avenue of inquiry.

Perhaps the most exciting and rapidly developing indication for endovascular techniques in the cerebral circulation is that of thrombolytic therapy for acute occlusive stroke. While trials of intravenous thrombolytic therapy are taking place at major medical centers, the ability to selectively administer thrombolytic and neuroprotective agents may render our present "treatment" of acute stroke obsolete. Direct intraarterial administration of urokinase, a thrombolytic agent, into the acutely occluded basilar and carotid vessels has had some success. The results of our limited experience using intraarterial tissue plasminogen activator to lyse thromboembolic material in the acute stages of stroke appear promising. Far more work needs to be done, but the benefits could be enormous.

Interventional endovascular therapy for cerebrovascular disease has already had important effects. Certain limitations exist, and open microsurgical techniques and stereotactic radiosurgery remain mainstays of treatment in the foreseeable future. Nonetheless, with further technical advances and clinical experience, endovascular therapy for disorders in the central nervous system can be expected to flourish.

Barton Lane, M.D.
Michael P. Marks, M.D.
Gary K. Steinberg, M.D., Ph.D.
Stanford, California

REFERENCES

Fox AJ, Pelz DM, Lee DH: Arteriovenous malformations of the brain: Recent results of endovascular therapy. *Radiology* 1990; 177: 51-7.

Guglielmi G, Vinuela F, Dion J, Duckwiler G: Electrothrombosis of saccular aneurysms via endovascular approach—Part 2: Preliminary clinical experience. *J Neurosurg* 1991; 75:8-14.

Marks MP, Lane B, Steinberg GK, Snipes GJ: Intranidal aneurysms in cerebral arteriovenous malformations: Evaluation and endovascular treatment. *Radiology* 1992; 183:355-60.

Newell DW, Eskridge JM, Mayberg MR, et al: Angioplasty for the treatment of symptomatic vasospasm following subarachnoid hemorrhage. *J Neurosurg* 1989; 71:654-60.

Preventive Aspects of Helmet Safety

HELMET use has been shown to be effective in reducing the extent of injury in both motorcycle and bicycle accidents. Motorcycle helmet use reduces the incidence of severe head injuries by 50%, regardless of the speed limit. After repeal of Kansas's helmet law, the incidence of head injuries increased by 70%, with helmetless motorcyclists at an 81% greater risk of serious injury. In 1990 bicycle helmet use became compulsory in Victoria, British Columbia. It resulted in a marked reduction in the number of hospital admissions of injured cyclists and in the proportion of head injuries compared with the previous year.

In 1974 Congress repealed the U.S. Secretary of Transportation's right to influence states to enforce helmet laws. Arguments to repeal mandatory helmet laws include an infringement on personal rights, the increased rate of accidents due to visibility and hearing limitations (not shown to be true), helmet weight contributing to the severity of injury (not shown to be true), and a lack of proof of effectiveness in preventing injury (not true). Full-coverage helmets have been shown to reduce horizontal peripheral vision by only 3%. Furthermore, a noise loud enough to be heard over the baseline noise of the motorcycle can easily be heard by a helmeted rider.

Research into the construction of helmets has resulted in improved effectiveness and comfort over the cork-lined helmets used during the World War II era. An effective helmet has a plastic or fiberglass rigid shell with a chin strap and an energy-absorbing foam liner. Most impacts to helmets in both motorcycle and bicycle accidents occur on the front and sides with infrequent penetration injuries.

Motorcycle helmet standards are reasonably consistent from country to country. In general the helmet should protect the victims from an impact velocity of 6 to 7 m per second or about 20 to 25 km per hour. These figures are reasonable because 75% of motorcycle injuries occur at less than 48 km per hour. Only in Great Britain is there a standard for faceguard or chin-strap impact testing. The protective properties of the face guard at other than low-velocity impacts remain controversial.

A stiffer shell results in diffusion of the impact and in a rebound of the energy transmission. The protective characteristics of the foam liner depend on the impact velocity. Stiffer foam is more effective at high velocities, whereas a less stiff foam provides more protection at low velocity. Increasing the thickness of the liner leads to a better level of protection at higher velocities but may also increase user discomfort due to the increased mass and weight of the helmet.

Standards for bicycle helmets changed in 1990. The penetration test was replaced by resistance testing to localized loading, thereby allowing the use of soft-shell helmets. In laboratory and field testing, the effectiveness of both the hard-shell and soft-shell helmets was comparable.

Researchers continually strive to construct helmets that offer optimum protection and comfort. The best helmet made is worthless if it sits on the shelf, however.

Theresa A. Hadden, R.N.
Edward C. Benzel, M.D.
Albuquerque, New Mexico

REFERENCES

Hopes PD, Chinn BP: Helmets: A new look at design and possible protection, *In* Proceedings of the International IRCOBI Conference of the Biomechanics of Impacts. Bron. France, International Research Council on the Biokinetics of Impacts, 1989, pp 39-54.

Ryan GA: Improving head protection for cyclists, motorcyclists, and car occupants. *World J Surg* 1992: 16:398-402.

Care of Patients With Aneurysms

THE survival of patients with untreated ruptured cerebral aneurysm is poor. By contrast, surgical treatment is clearly beneficial. It is imperative, however, to make the diagnosis of subarachnoid hemorrhage as soon as possible. The initial bleeding event is often missed. It can be mild, but a subsequent hemorrhage is often devastating if not fatal. Middleaged patients who present with a severe headache, frequently described as "the worst headache of my life," with or without neck stiffness must be suspected of having subarachnoid hemorrhage, particularly those with hypertension or atherosclerosis. Many patients are misdiagnosed as having flu or other viral illness, migraine (which only rarely starts late in life), or tension headache. Lumbar puncture, computed tomographic (CT) scan, or both will settle the issue in most cases. More severe subarachnoid hemorrhage resulting in mental obtundation with or without neurologic deficit is not usually a diagnostic problem.

The treatment of subarachnoid hemorrhage is surgical in most cases. Traditionally the operation has been done after a waiting period of two weeks to allow the swelling and vasospasm to resolve. The outlook for those patients who improved during that two-week period was excellent. Overall management results, however, indicate that 60% of the patients die or are disabled as a result of rebleeding or vasospasm during that period of waiting. Numerous therapeutic strategies have been developed to manage these complications, including early surgical intervention.

Prospective studies indicate that rebleeding occurs in more than 20% of patients in the first two weeks after the initial bleeding event, with a peak incidence in the first 24 to 48 hours. Mortality associated with a second hemorrhage is high (70% to 80%). Only obliteration of the aneurysmal sac can predictably avert this occurrence. Antifibrinolytic agents, such as aminocaproic acid (Amicar), reduce the incidence of rebleeding but increase the incidence of ischemia and stroke. Induced hypotension has similar deleterious effects. Ischemic deficits from vasospasm develop in a third of patients surviving the initial bleeding event and leave them disabled. While the exact pathophysiology of vasospasm is unclear, the amount of blood in the subarachnoid cisterns predicts the severity of the vasospasm. Induced hypervolemia and hypertension, as well as the use of calcium channel blockers, are now routinely used to combat the effects of vasospasm, but success is only modest. Transcranial Doppler studies and single photon-emission CT scanning can be useful guides to the evolution and severity of vasospasm.

With recent advances in neuroanesthesia, pharmacologic cerebral protection, intraoperative monitoring, and microsurgical instrumentation and techniques, neurosurgeons at a number of centers are turning to early surgical interventions. This is an effort to avert death from spontaneous rebleeding and to protect against rebleeding as a complication of the induced hypertension and hypervolemia used to treat vasospasm. Furthermore, preliminary experience with angioplasty, which can help ameliorate vasospasm in selected patients, indicates that it may be safer following aneurysm obliteration.

Several reports, including the International Cooperative Study on the timing of aneurysm surgery, have documented good results from early surgical intervention. There appears to be an advantage to early surgical treatment in patients who are in good neurologic condition on admission. There are no compelling data on poorer grade patients to recommend early operation, but anecdotal reports suggest that early surgical treatment and aggressive medical management may yield a better result in these patients as well. Other benefits of this treatment include the early removal of intracerebral hematomas, fewer medical complications, probably a lowered incidence of the hydrocephalus that may follow subarachnoid hemorrhage, less psychological stress for the patient and the family, and probably a shorter hospital stay. A somewhat higher operative morbidity and mortality must be accepted for early surgical treatment compared with traditional delayed operation to realize the benefits in total management morbidity and mortality.

Peter Le Roux, M.D.
H. Richard Winn, M.D.
Seattle, Washington

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

REFERENCES

Bailes JE, Spetzler RS, Hadley MN, Baldwin HZ: Management morbidity, and mortality of poor-grade aneurysm patients. *J Neurosurg* 1990; 72:559-96.

Haley EC, Kassell NF, Torner JC, and the Participants: The International cooperative study on the timing of aneurysm surgery: The North American experience. *Stroke* 1992; 23:205-14.

Saveland H, Hillman J, Brandt L, et al: Overall outcome in aneurysmal and subarachnoid hemorrhage—A prospective study from neurosurgical units in Sweden during a one-year period. *J Neurosurg* 1992; 76:729-34.

Winn HR, Newell DW, Mayberg MR, et al: Early surgical management of poor-grade patients with intracranial aneurysms. *Clin Neurosurg* 1990; 36:289-98.

Epilepsy Surgery for Hippocampal Sclerosis

HIPPOCAMPAL sclerosis is the most frequent pathologic abnormality associated with medically intractable temporal lobe epilepsy. In fact, it is seen in a third of all cases in a random population of patients suffering from epilepsy, as reported both from autopsy cases and in histologic studies on temporal lobes removed during an operation for epilepsy. Hippocampal sclerosis indicates shrinkage and induration of specific regions of the hippocampus with histologic evidence of neuronal loss and secondary astroglial fiber proliferation. There is also a loss of inhibitory hippocampal interneurons and evidence of synaptic reorganization. Hippocampal sclerosis can be either unilateral or bilateral; in some autopsy series, more than half the patients with hippocampal sclerosis had bilateral lesions.

Patients in whom epilepsy manifests itself early seem to show evidence of hippocampal sclerosis more often than patients with late-onset seizures. Bilateral symmetric hippocampal sclerosis is associated with an early onset. The duration of epilepsy, however, does not correlate with the incidence of hippocampal sclerosis. Unilateral, predominantly selective, hippocampal sclerosis is seen in patients with epilepsy beginning later in life. Hippocampal sclerosis can also be seen in infantile spasms and in the Lennox-Gastaut syndrome.

Before any patient with seizures can become a surgical candidate, the patient must undergo a complete evaluation. Patients must be under the care of a neurologist and have seizures that are not controlled with appropriate anticonvulsant therapy. The history and physical examination, scalp interictal and ictal electroencephalograms, magnetic resonance imaging (MRI), and neuropsychological testing are all done to localize the region of epileptogenesis. If the site is localized, an intracarotid sodium amobarbital test should be done to identify the hemispheric distribution of language and memory. If memory cannot be supported by the temporal lobe contralateral to the proposed resection, then the patient is not a candidate for temporal lobe resection.

Magnetic resonance imaging findings help lateralize the seizure focus by showing hippocampal asymmetry. Hippocampal sclerosis may be detected as atrophy of the mesial temporal structures on T1-weighted images and increased signal intensity on T2-weighted images. Other MRI findings, such as atrophy of the adjacent temporal lobe and temporal horn dilatation, may be present. Computed tomographic scanning is rarely helpful because of the relatively poor resolution of the mesial temporal structures. Positron emission tomography may help define regions of functional abnormality, usually hypometabolism, in the suspected temporal lobe.

If the epileptic region is not sufficiently localized to proceed with surgical therapy after the noninvasive evaluation, then intracranial electrodes can be surgically implanted. To define an epileptogenic region thought to be in the limbic cortex, depth electrodes can be implanted in the amygdala and hippocampus through either the inferior or middle temporal gyrus or the occipital lobe. Subdural strip electrodes can also be placed against the inferior and mesial temporal cortex. Recording from lateral temporal or extratemporal neocortex is usually accomplished with subdural strip or grid electrodes placed over the area of interest.

The operation most often done is removal of the temporal lobe en bloc. The procedure should include resection of the amygdala and hippocampus. Depending on the presence of language function in the temporal lobe being removed, as well as the results of the electrical recordings done on the patient, 3.5 to 5.5 cm of lateral temporal lobe measured from the anterior pole is removed. The lateral extent of the resection may be less extensive than the mesial extent.

With this approach, the outcome for these patients is good. After the operation at least 70% of patients are seizure-free with medication or have only auras, 20% have more than 80% reduction in seizure frequency, and 10% have less than 80% reduction in seizure frequency. Most patients who are benefited by surgical treatment will be able to reduce the amount of anticonvulsants that they require, giving them an increased functional capacity. Morbidity from the various surgical procedures associated with epilepsy, such as hematomas or infections, is low, and operative mortality is rare.

Conrad T.E. Pappas, M.D., Ph.D.
Davis, California

REFERENCES

Chung MY, Walczak TS, Lewis DV, et al: Temporal lobectomy and independent bitemporal interictal activity: What degree of lateralization is sufficient? *Epilepsia* 1991; 32:195-201.

Primrose DC, Ojemann G: Outcome of resective surgery for temporal lobe epilepsy, chap 67, *In* Luders HO (ed): Epilepsy Surgery. New York, NY, Raven Press, 1992, pp 601-11.

Spencer DD, Inserni J: Temporal lobectomy, chap 61, *In* Luders HO (ed): Epilepsy Surgery. New York, NY, Raven Press, 1992, pp 533-4.5

Approaches to Cervical Myeloradiculopathy

CERVICAL myeloradiculopathy is a common cause of radicular pain and weakness in the arms and progressive leg weakness, spasticity, and gait disturbance in the later decades of life. There are two major presenting syndromes of the myelopathy. The first is the transverse syndrome that presents with spastic legs, disturbances of gait, and loss of joint position sense and vibratory sensation in the lower extremities. Individual patients will have varying degrees of severity of the different components. The second syndrome is called the central syndrome and is less common. Involvement is primarily in the upper extremities, where patients will show varying degrees of weakness and sensory loss. There is typically major involvement of the hands with weakness and atrophy of intrinsic hand muscles and sensory loss often associated with dysesthesias. Bladder dysfunction may also be seen. Combinations of the two syndromes are not uncommon.

Degenerative spondylotic myeloradiculopathy is thought to be the result of chronic, repetitive compressive damage to the cervical spinal cord and roots. Compression may result primarily from anterior spondylotic spurs, posterior infolding of the ligamenta flava, or both. When the myelopathic spinal cord is examined, changes are found that suggest chronic demyelination, vascular compromise, and inflammation of the nerve roots. The exact relationship of this disorder to compressive forces is not fully delineated.

When only one or two levels of anterior spondylotic compression are recognized, anterior cervical discectomy with or without fusion for the removal of the offending osteophytes achieves excellent results. If more than one or two levels of anterior compression are found, then posterior cervical laminectomy has formerly been advocated because of the perceived inability to deal surgically with multilevel anterior disease.

The treatment of cervical spondylotic myeloradiculopathy has changed recently. Surgical approaches are aimed at removing the offending compressive disorder, anterior or posterior, rather than just decompressing the spinal cord so that it can move into a better "position." When multilevel anterior involvement is identified, aggressive anterior approaches, including the partial removal of two or more vertebral bodies, the posterior longitudinal ligament, and any remaining lateral spurs, allows a successful decompression of three or more disc spaces and associated nerve roots. The use of anterior cervical plates has advanced these approaches because the plates serve to anchor and stabilize the grafts and in many cases eliminate the necessity for halo orthosis. The results of aggressive anterior approaches have been excellent, improving on the results of simple posterior decompression alone.

At the same time that aggressive anterior decompression has become more common, simple posterior laminectomy remains an important technique. Laminectomy is indicated for patients with one or two levels of cervical stenosis and root compression, primarily caused by posterior compressive processes, and for those with multilevel disease that cannot be adequately addressed anteriorly. The results of cervical laminectomy compare favorably with those of anterior cervical decompression in these patients.

In the orthopedic literature, there is interest in cervical laminoplasty. This technique, developed in Japan for the treatment of ossification of the posterior longitudinal ligament, seeks to decompress the spinal cord posteriorly and to reconstruct an enlarged cervical spinal canal with intact laminae. By maintaining the integrity of the posterior spinal elements, the potential for postoperative cervical kyphosis and instability is thought to be limited. The results of cervical laminoplasty for cervical spondylotic disease are currently being investigated.

With the introduction of newer anterior and posterior approaches to cervical disorders producing myeloradiculopathies, a careful analysis of cervical compressive pathophysiology is necessary so that appropriate therapy is initiated.

Edmund Frank, M.D.
Portland, Oregon

REFERENCES

Bertalanffy H, Eggert HR: Clinical long-term results of anterior discectomy without fusion for treatment of cervical radiculopathy and myelopathy: A follow-up of 164 cases. *Acta Neurochir* (Wien) 1988; 90:127-35.

Herkowitz HN: A comparison of anterior cervical fusion, cervical laminectomy and cervical laminoplasty for the surgical management of multiple level spondylitic radiculopathy. *Spine* 1988; 13:774-80.

Okada K, Shirasaki N, Hayashi H, et al: Treatment of cervical spondylitic myelopathy by enlargement of the spinal canal anteriorly, followed by arthrodesis. *J Bone Joint Surg* [Am] 1991; 73:352-64.

Saunders RL, Bernini PM, Shirreffs TG Jr, Reeves AG: Central corpectomy for cervical spondylotic myelopathy: A consecutive series with long-term follow-up evaluation. *J Neurosurg* 1991; 74: 163-70.

Seifert V, Stolke D: Multi-segmental cervical spondylosis: Treatment by spondylectomy, microsurgical decompression, and osteosynthesis. *Neurosurgery* 1991; 29:498-503.

Tomita K, Normura S, Umeda S, Baba H: Cervical laminoplasty to enlarge the spinal canal in multilevel ossification of the posterior longitudinal ligament with myelopathy. *Arch Orthop Trauma Surg* 1988; 107:148-53.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Metastatic Spinal Tumors

METASTATIC spinal tumors are common and potentially devastating neurosurgical emergencies. Irreversible paraplegia or quadriplegia may develop rapidly without much warning. Deficits may progress over hours if early symptoms are ignored by the patient or if the diagnosis is delayed. As many as 50% of patients can no longer walk at the time treatment is initiated.

Spinal tumors are metastatic in 89% of cases. The vertebral column is the third most prevalent site of metastatic disease. Spinal metastases are found in 20% to 70% of patients with malignant neoplasms; bronchogenic, breast, prostate, and hematopoietic sources are the most common. Spinal cord compression results in 25% of cases where tumors metastasize to the vertebral column. Neurologic deficit due to vertebral lesions is the presenting symptom in 8% to 14% of cancer patients. The median survival after the onset of spinal cord compression is reported to be as long as 12 to 18 months depending on the tumor cell type, the extent of the primary and metastatic disease, the age of the patient, the severity or rate of progression of the neurologic deficit, and the location or extent of the vertebral neoplasm.

The initial symptom is localized spinal pain and tenderness. Radicular radiation is more common with cervical or lumbar lesions than with thoracic lesions. Spinal pain in a patient with known or suspected malignant disease demands prompt radiographic studies. Numbness and demonstrable sensory deficit beginning two to four dermatomal segments below the site of pain and either preceded or followed by impairment of leg coordination, manual dexterity, or bladder control herald the onset of spinal cord or cauda equina compression. For therapeutic purposes, patients are best classified as asymptomatic, symptomatic but ambulatory, or nonambulatory with complete or incomplete deficits.

Advances in methods for diagnostic magnetic resonance imaging, image-guided needle biopsy, preoperative endovascular embolization for hemostasis, microsurgery for anterior spinal cord decompression, and various instrumentation techniques for spinal stabilization have initiated a quiet revolution in the management of vertebral metastases. Magnetic resonance imaging is the best technique for diagnosing spinal metastases. The entire spinal column can be shown in the sagittal plane, and axial images through the involved segments show epidural mass effect from tumor invasion. The ability to identify the exact extent, site, and cause of spinal cord and nerve root compression or vertebral instability early in the course of metastatic disease has dramatically improved the results of both radiation and surgical therapy.

Mild stable or slowly progressive neurologic deficits caused by radiosensitive tumors are best treated by steroids and radiation therapy. Rapidly progressive but incomplete neurologic deficits require immediate surgical decompression and stabilization if instability exists. Another spinal complication of metastatic disease that may require surgical attention is pathologic vertebral fracture. Displaced bone fragments in the spinal canal may cause spinal cord compression with or without instability. Subluxation of vertebral bodies, which may cause or aggravate spinal cord compression, may occur when more than 50% of a vertebral body is destroyed, when there is both anterior and posterior vertebral involvement or bilateral posterior involvement, or when there is extensive multilevel disease.

Because as many as 85% of spinal metastases are anterior, the traditional surgical approach of laminectomy offers no better results than radiation therapy (32% to 38% ambulatory after treatment) and is rarely indicated. Anterior cervical, anterior, or posterolateral thoracic and lateral extracavitary thoracolumbar and lumbar approaches for vertebrectomy, radical decompression of the neural elements, and internal fixation relieve pain in 81% to 94% of patients and restore ambulation in 75% of patients with incomplete motor deficits. Vertebral body replacement with acrylic and wire or metallic fixation devices rather than bone grafting is adequate to correct kyphosis and maintain stability for the expected duration of patient survival. Posterior fixation with pedicle screws or rods and sublaminar wires at levels above and below the tumor may also be necessary. With aggressive management of spinal metastases, the quality of survival in patients with malignant disease may be considerably enhanced despite the risks of an operation.

Philip R. Weinstein, M.D.
San Francisco, California

REFERENCES

Harrington KD: Anterior cord decompression and spinal stabilization for patients with metastatic lesions of the spine. *J Neurosurg* 1984; 61:107-7.

Schaberg J, Gainor BJ: A profile of metastatic carcinoma of the spine. *Spine* 1985; 10: 19-20.

Siegal T, Siegal T: Surgical decompression of anterior and posterior malignant epidural tumors compressing the spinal cord—A prospective study. *Neurosurgery* 1985; 17:424-30.

Sundaresan N, Digiacinto GV, Hughes JE, et al: Treatment of neoplastic spinal cord compression: Results of a prospective study. *Neurosurgery* 1991; 29:645-50.

Sundaresan N, Krol G, Digiacinto G, Hughes JE: Metastatic tumors of the spine, chap 29 *In* Sundaresan N Schmidek HH, Schiller AL, Rosenthal Dl (eds): Tumors of the Spine: Diagnosis and Clinical Management. Philadelphia, Pa, WB Saunders, 1990, pp 279-304.

ADVISORY PANEL TO THE SECTION ON NEUROSURGERY

Sidney Tolchin, M.D.
Advisory Panel Chair
CMA Council on Scientific Affairs Representative
La Mesa

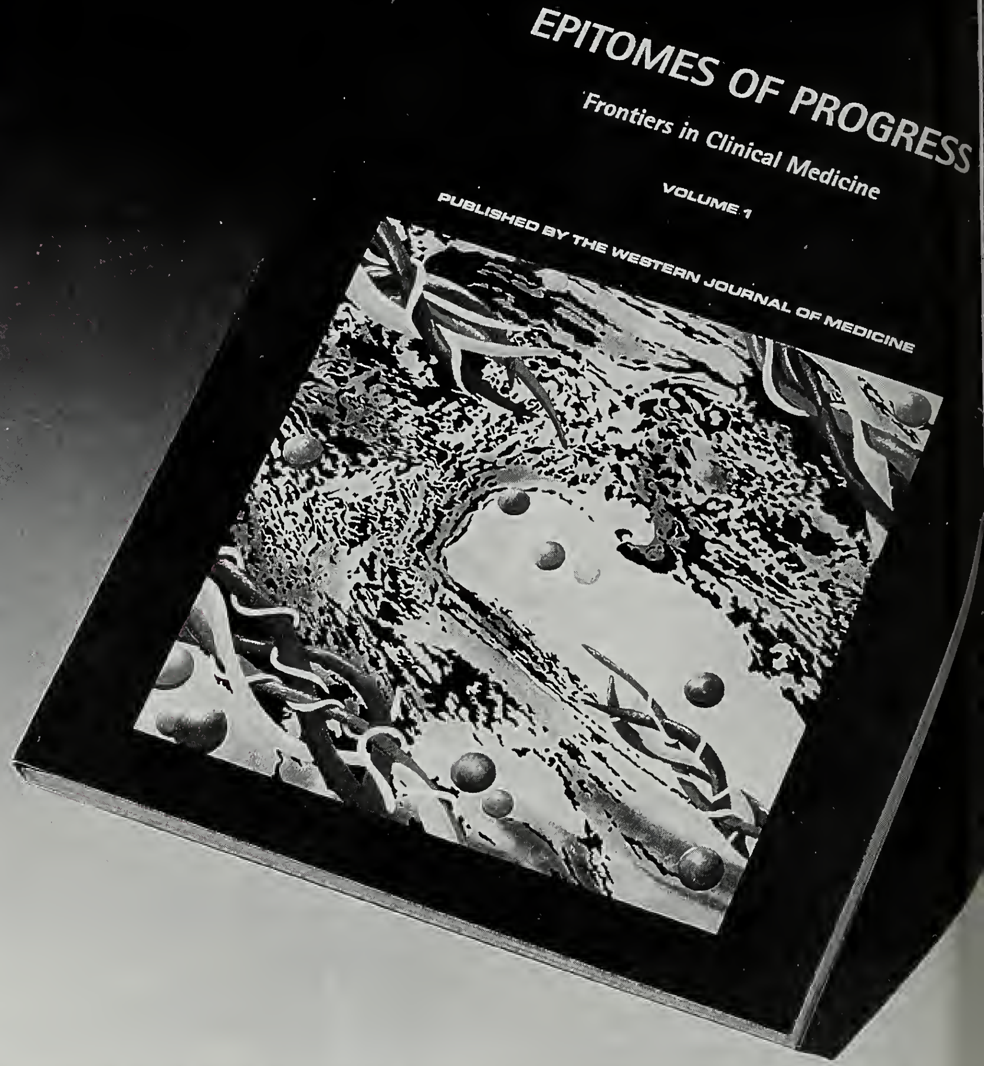
Michael Sukoff, M.D.
Santa Ana
Peter Dyck, M.D.
Los Angeles
Lawrence M. Shuer, M.D.
Stanford University
Shokei Yamada, M.D.
Loma Linda University
Franklin C. Wagner, Jr., M.D.
University of California
Davis
Ronald F. Young, M.D.
University of California
Irvine

Donald P. Becker, M.D.
University of California
Los Angeles
John F. Alksne, M.D.
University of California
San Diego
Charles B. Wilson, M.D.
University of California
San Francisco
Martin Weiss, M.D.
University of Southern California
Ulrich Batzdorf, M.D.
California Association of
Neurological Surgeons
Los Angeles
Morris D. Loffman, M.D.
California Association of
Neurological Surgeons
Van Nuys

George H. Koenig, M.D.
California Association of
Neurological Surgeons
Redwood City
Gerald Silverberg, M.D.
Section Editor
California Association of
Neurological Surgeons
Stanford
Philipp M. Lippe, M.D.
California Association of
Neurological Surgeons
San Jose

epitome

i-ˈpit-ə-mē\ *n* [L, fr. Gk *epitomē,* fr. *epitemnein* to cut short, fr. *epi-* + *temnein* to cut — more at TOME] (1520) **1 a** : a summary of a written work **b** : a brief presentation or statement of something **2** : a typical or ideal example: EMBODIMENT (the British monarchy itself is the ~ of tradition — Richard Joseph) **3** : Plural — *Epit-o-mes* — A regular FEATURE of *The Western Journal of Medicine*, now available in a single, bound volume.


EPITOMES OF PROGRESS
Frontiers in Clinical Medicine
VOLUME 1
PUBLISHED BY THE WESTERN JOURNAL OF MEDICINE

Each set of **EPITOMES** is a concise and clear review of the important advances in a medical specialty, chosen and written by experts in that specialty. Now, for the first time, a special bound volume of **EPITOMES** is available. For only $19.95 you can have all the latest advances in 24 different specialties in one attractively designed volume. An excellent reference for your library. A useful gift. Hurry! Supplies are limited. Order a copy today.

YES, SEND A SPECIAL BOUND EDITION OF **EPITOMES** TO:

The Western Journal of Medicine

☐ Yes, send me _____ copy(ies) of the Epitomes book at $19.95 each.

☐ I want to receive Epitomes every month. Please enter my one-year subscription to *WJM* for only $40.00.

For additional information, call (415) 882-5179.
Please mail this coupon with enclosed payment to:
Circulation Department
P.O. Box 7602, San Francisco, California 94120-7602

Name ______________________
Street ______________________
City ______________________
State ______________ Zip ______________
☐ Check enclosed Charge my: ☐ VISA ☐ MasterCard
Card Number ______________________
Expiration Date ______________________
Signature ______________________

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Choice of Cholesterol-lowering Drugs

THE MEDICAL LETTER

LOWERING elevated serum cholesterol concentrations can slow progression and sometimes cause regression of atherosclerotic lesions. Most authorities advise patients with high cholesterol concentrations to eat less fat and less cholesterol and, when appropriate, to lose weight. If these measures do not lower serum lipids sufficiently, drugs are frequently added to the regimen. When drugs are discontinued, serum cholesterol concentrations generally return to pretreatment levels.

LIPOPROTEINS, TRIGLYCERIDES, AND APOLIPOPROTEINS—Most of the cholesterol in plasma is carried in low-density lipoproteins (LDL); high serum concentrations of LDL are associated with an increased risk of coronary heart disease. High serum concentrations of high-density lipoproteins (HDL) are associated with a low incidence of coronary heart disease. Triglycerides are carried in the plasma mainly in very-low-density lipoproteins (VLDL); they may also play a role in the pathogenesis of coronary atherosclerosis (NIH Consensus Development Panel, *JAMA*, 269:505, Jan 27, 1993). Apolipoproteins are protein constituents of lipoproteins. High concentrations of apolipoprotein B (the main structural protein of LDL and VLDL) and of lipoprotein(a) have been associated with an increased risk of coronary artery disease, but laboratory determinations are not widely available (Scanu AM, et al: *Ann Intern Med* 1991; 115:209; Genest J, Jr., et al, *J Am Coll Cardiol* 1992; 19:792).

CHOLESTYRAMINE AND COLESTIPOL—The bile acid-binding resins cholestyramine *(Questran; Questran Light)* and colestipol *(Colestid)* lower concentrations of LDL cholesterol. A seven- to 10-year double-blind randomized trial compared the effects of cholestyramine with placebo in 3,806 men, 35 to 59 years old, free of coronary disease but with a baseline plasma cholesterol of at least 265 mg/dl (6.87 mmol/L—the 95th percentile for men in their forties); all patients followed a moderate cholesterol-lowering diet. After seven years, the incidence of nonfatal myocardial infarction or death from coronary heart disease was 8.6% in the placebo-treated patients and 7% in the cholestyramine group; overall mortality was not affected (The Lipid Research Clinics Primary Prevention Trial, *JAMA* 1984; 251:351, 365). In an angiographic study, 90 men with coronary heart disease were assigned at random to usual care, diet, or diet plus cholestyramine; after 39 months, both diet and diet plus cholestyramine decreased the percentage of patients with progressive coronary narrowing and increased the percentage with coronary widening; the increase in the coronary lumen was greater with cholestyramine (Watts GF, et al: *Lancet* 1992; 339:563).

Adverse Effects—The adverse effects of colestipol and cholestyramine are similar; constipation occurs frequently and may be accompanied by heartburn, nausea, belching, and bloating, but these tend to disappear over time. Giving the drug in moderate doses (eight to 10 grams daily) just before meals improves tolerance, and increased dietary fiber or a fiber supplement, such as psyllium *(Metamucil,* and others), may relieve constipation and bloating. Both cholestyramine and colestipol may interfere with absorption of fat-soluble vitamins and many other drugs *(The Medical Letter Handbook of Adverse Drug Interactions* 1993; pp. 81, 91). Levels of triglycerides may rise, especially in patients with hypertriglyceridemia.

NIACIN (NICOTINIC ACID)—Although the mechanism is not known, niacin decreases triglycerides, total serum cholesterol and LDL cholesterol, and increases HDL cholesterol. It also lowers lipoprotein(a), except in patients with severe hypertriglyceridemia. In one placebo-controlled trial, men with one or more previous myocardial infarctions who took niacin had a lower incidence of nonfatal myocardial infarction and, nine years later, a lower mortality rate (Canner PL, et al: *J Am Coll Cardiol* 1986 8:1245).

Adverse Effects—Niacin can cause flushing, pruritus, and gastrointestinal distress, but these problems can be relieved by taking aspirin before each dose and can be minimized by taking a low initial dosage, increasing it

---

Reprinted from *The Medical Letter*, Vol. 35, 5 March 1993.

gradually, and taking the drug with meals. Niacin can also cause blurred vision, glucose intolerance, hyperuricemia, and, rarely, dry eyes or hyperpigmentation. Hepatic toxicity, sometimes severe, has been reported with both regular and, more frequently, sustained-release preparations (Henkin Y, et al: *JAMA* 1990; 264:241; Etchason JA, et al: *Mayo Clin Proc* 1991; 66:23; Frost PH: *Ann Intern Med* 1991; 114:1065).

HMG-CoA REDUCTASE INHIBITORS—Pravastatin *(Pravachol—*Bristol-Myers Squibb) and simvastatin *(Zocor—*Merck), two new inhibitors of cholesterol synthesis (*Medical Letter* 1992; 34:57), are similar to lovastatin *(Mevacor—*Merck).

Effect on Serum Cholesterol—All of these drugs inhibit HMG-CoA (3-hydroxy-3-methylglutaryl-coenzyme A) reductase, the enzyme that catalyzes the rate-limiting step in cholesterol synthesis; they are more effective than other types of drugs in lowering plasma concentrations of low-density-lipoprotein (LDL) and total cholesterol. Few direct comparisons of the HMG-CoA reductase inhibitors have been published. In one study, 20 mg of simvastatin was about as effective in lowering total cholesterol as 40 mg of lovastatin (Illingworth DR, et al: *Atherosclerosis* 1992; 96:53). Another study found 20 mg of lovastatin as effective as 20 mg of pravastatin (McPherson R, et al: *Clin Ther* 1992; 14:275). In a third study, 10 mg of simvastatin was more effective in lowering plasma lipids than 10 mg of pravastatin (European Study Group, *Am J Cardiol* 15 Nov 1992; 70:1281). When these drugs are taken once a day, it should be in the evening because most cholesterol synthesis occurs between midnight and 3 AM; their effectiveness decreases when taken in the morning.

Effect on Coronary Atherosclerosis—A controlled angiographic trial in 270 patients on a low-fat diet found that lovastatin, 80 mg per day, caused less progression and more regression of atherosclerotic lesions than placebo (Blankenhorn, D, et al: presented at 59th Congress of European Atherosclerosis Society, Nice, 17-21 May 1992). Similar studies are not available for pravastatin or simvastatin.

Adverse Effects—All three drugs are generally better tolerated than other cholesterol-lowering drugs (*Medical Letter* 1991; 33:1). Mild, transient gastrointestinal disturbances, rash, and headache have been the most common adverse effects, and some patients have reported sleep disturbances. An increase in plasma aminotransferase activities to more than three times normal occurs in one percent to two percent of patients taking higher doses of these drugs and may require discontinuation, but symptomatic hepatitis has been rare. A lupus-like syndrome has been reported with both lovastatin and simvastatin (Ahmad S, *Arch Intern Med* 1991; 151:1667; Bannwarth B, et al: *Arch Intern Med* 1992; 152:1093).

Myopathy—Severe myalgia with more than ten-fold increases in serum creatine phosphokinase concentrations has been reported rarely with all three drugs. Hepatic disease, severe infections, renal failure, and concurrent treatment with cyclosporine *(Sandimmune),* gemfibrozil *(Lopid),* niacin, or erythromycin have increased the incidence of myopathy. Rarely, myoglobinuria has led to acute renal failure.

Long-term—Cholesterol itself is a component of all human cell membranes; the long-term consequences of interfering indefinitely with its synthesis and the synthesis of related compounds are unknown. Hepatocellular cancer and lymphoma have been reported in animals treated with high doses of an HMG-CoA reductase inhibitor.

GEMFIBROZIL—A fibric acid derivative that decreases synthesis of apolipoprotein B, gemfibrozil *(Lopid)* lowers VLDL and can increase HDL cholesterol. It is also highly effective in lowering plasma triglycerides, but when the triglyceride concentration decreases, LDL cholesterol may increase in some patients. A five-year double-blind trial (the Helsinki Heart Study) in 4,081 asymptomatic men with hypercholesterolemia showed not only decreased triglycerides, decreased LDL cholesterol, and increased HDL cholesterol in men treated with gemfibrozil, but also a decrease in the number of myocardial infarctions (56 compared to 84 with placebo) (Manninen V, et al: *JAMA* 1988; 260:641). After three point five years of additional follow-up, however, the total number of deaths was 83 in patients originally given placebo and 101 in those originally treated with gemfibrozil; after the five-year trial, some patients in the placebo group took gemfibrozil, and some patients in the gemfibrozil group took nothing (Newman TB, Canter D, Perlow L: *N Engl J Med* 14 Jan 1993; 328:139).

Adverse Effects—In the Helsinki Heart Study, only the incidence of upper gastrointestinal symptoms was slightly increased in men taking gemfibrozil. The drug may potentiate the effect of oral anticoagulants and also increases biliary cholesterol saturation, which may increase formation of cholesterol gallstones.

CLOFIBRATE—Clofibrate *(Atromid-S;* and others) is a fibric acid derivative similar to gemfibrozil. During treatment with clofibrate, 5,000 hypercholesterolemic men had an increased rate of mortality due to malignant and gastrointestinal disease, and a higher rate of cholecystectomy for gallstones (Heady JA, et al: *Lancet* 5 Dec 1992; 340:1405). Its use should probably be limited to patients with severe hypertriglyceridemia unresponsive to gemfibrozil.

PROBUCOL—Probucol *(Lorelco)* generally lowers total cholesterol and LDL cholesterol by 10% to 20% in responding patients, but some do not respond and the drug

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

also lowers HDL cholesterol by 20% to 30%. Probucol inhibits oxidation of LDL, however, and decreases its uptake by macrophages, which theoretically could inhibit atherogenesis without decreasing serum cholesterol concentrations (Steinberg D, Witztum JL: *JAMA* 1990; 264:3047). It has caused regression of xanthomas in patients with homozygous familial hypercholesterolemia (Yamamoto A, et al: *Am J Cardiol* 1986; 57:29H).

Adverse Effects—Probucol has a long half-life and is released from fat stores for months after treatment has stopped. It is generally well tolerated, but occasionally causes loose stools and diarrhea, and may prolong the QT-interval.

FISH OIL—Large amounts of long-chain, highly unsaturated omega-3 fatty acids (present in cold water fish and commercially available in capsules) can decrease triglycerides and cause small decreases (or sometimes increases) in LDL cholesterol, without affecting HDL cholesterol (*Medical Letter* 1987; 29:7). No studies are available comparing the effectiveness or safety of fish oil with other lipid-lowering drugs. A two-year study, however, in male survivors of myocardial infarction, showed that a moderate intake of fatty fish or fish oil decreased total mortality by 29%; this effect occurred without any reduction in serum cholesterol levels (Burr ML, et al: *Lancet* 1989; 2:757). Omega-3 fatty acids from fish can interfere with platelet function, but bleeding has not been reported.

COMBINATIONS—When tolerable doses of a single drug do not lower blood lipids sufficiently, two drugs can be used together. One study compared the combination of niacin plus colestipol with placebo in men with previous coronary bypass surgery; after four years, those taking the active drugs had less progression of old atheromata, fewer new ones, and more regression of atherosclerosis in both native arteries and grafts (Cashin-Hemphill L, et al, *JAMA* 1990; 264:3013). In another angiographic study, patients taking colestipol combined with either niacin or lovastatin had less progression and more regression of coronary lesions after two and a half years than patients treated with diet alone or diet plus colestipol (Brown G, et al: *N Engl J Med* 1990; 323:1289). Concurrent use of niacin and colestipol has been associated with decreased serum concentrations of thyroxine (T4) and thyroxine-binding globulin, but patients have remained euthyroid. Taking a combination of an HMG-CoA reductase inhibitor and gemfibrozil can lower total cholesterol and increase HDL, but the effect on coronary artery disease of raising HDL is unknown, and use of the two drugs together increases the risk of severe myopathy (Kreisberg RA: *Am J Med* 1 Jan 1993; 94:1; Wiklund O, et al: *Am J Med* 1 Jan 1993; 94:13)

CONCLUSION—Cholestyramine, colestipol, and niacin have proven effective for lowering elevated serum cholesterol concentrations; most experts recommend trying them first, either individually or using one of the resins and niacin concurrently. Many clinicians, however, particularly in patients who have severe hypercholesterolemia or have had a myocardial infarction, begin with an HMG-CoA reductase inhibitor such as lovastatin, pravastatin, or simvastatin, because they are generally more effective and better tolerated.

Cost of Some Cholesterol-lowering Drugs

Drug	**Daily dosage**	**Cost***
Cholestyramine—Questran, Questran Light (Bristol Labs)	16 grams resin, divided	$91.20
Colestipol—Colestid (Upjohn)	20 grams, divided	79.31
Gemfibrozil—Lopid (Parke-Davis)	1200 mg, in 2 doses	60.20
Lovastatin—Mevacor (Merck)	20 mg once	57 .60
Niacin—average generic price	1 gram tid	6.44
Niacor (Upsher-Smith)		23.04
Nicolar(Rhone-Poulenc Rorer)		88.13
Pravastatin—*Pravachol* (Bristol-Myers Squibb)	20 mg once	49.91
Probucol—*Lorelco* (Marion Merrell Dow)	500 mg bid	61.56
Simvastatin—Zocor(Merck)	10 mg once	51.94

*Cost to the pharmacist for 30 days' treatment based on Average Wholesale Price listings in *Drug Topics Red Book* 1992 and *Update* February 1993.

ENDORSED BY

THE MEDICAL SOCIETY OF NEW JERSEY

**V.E.B.A. PLAN*

FEATURES

TAX DEDUCTIBLE CONTRIBUTIONS TO AN I.R.S. APPROVED TRUST

EVEN IF . . .

- **YOUR PENSION PLAN IS MAXIMUM FUNDED**
- **YOUR PENSION PLAN IS OVER FUNDED**
- **YOU HAVE NO PENSION PLAN**

PLEASE SEND ME A copy of your informational pamphlet on the *V.E.B.A. program. I understand there is no obligation.

NAME ____

ADDRESS ____

DATE OF BIRTH ____________________ TELEPHONE # (_____)____________________

Mail to:

THE KIRWAN COMPANIES
402 MIDDLETOWN BLVD., SUITE 202
LANGHORNE, PA. 19047

(800) 283-7666...................... (215) 750-7616...................... FAX (215) 750-7791

*VOLUNTARY EMPLOYEE BENEFICIARIES ASSOCIATION

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

The Connecticut State Medical Society
and the
Connecticut Medical Insurance Company

W. RAYMOND JAMES, M.D.

THIS is the last of a series in three articles concerned with the history of Connecticut State Medical Society's involvement in the rapidly proliferating challenges of professional liability and malpractice. The first covered the years from the founding of the Connecticut State Medical Society in 1792 (including the first "malpractice" case in the United States in 1794) through 1969. The second covered the years from 1970, when the Society negotiated a new and broadened agreement with a professional liability company, through 1983 and the early planning for a physician-owned professional liability insurance company. While the Society is engaged in many activities aimed at improving the quality of patient care and reducing the cost to physician and patient of increasing compensable incidents and litigation, this article focuses on the organization and early operation of that company.

The CNA professional liability insurance plan described in the second article (with heavily discounted premiums the first year) had been widely accepted by CSMS membership since it was endorsed in 1981. However, the expected rapid rise in premiums, nearly doubling in three years, was unacceptably high. Accordingly, a special meeting of the CSMS Council was held 18 July 1984 for the "sole purpose of determining the society's action pertaining to the several professional liability options available." The Council had received materials representing a claims-made proposal from the St. Paul Insurance Company, a renewal offer from CNA with rate increases of approximately 41% for 1984, and an alternative proposal by Mr. John Dorsett of American Health Systems (AHS) to launch the captive company shelved in 1981. It was voted "that the Society immediately activate the Connecticut Medical Insurance Company (CMIC), to contract with AHS to implement and operate the new company, and that the Council chairman immediately appoint an Ad Hoc Committee to work with AHS."

Mr. Dorsett was expected to make a recommendation by 17 August as to whether the new company could successfully begin operation by October. He estimated the costs incurred by 17 August would not exceed 80,000 dollars. The council chairman, Dr. Guy Van Syckle, then appointed a seven-member committee as voted.* In the meantime AHS had made some changes and was known as American Insurance Systems (AIS), Mr. Bartholomew Nyhan largely replacing Mr. John Dorsett.

The Ad Hoc Committee met at least weekly with AIS officials, counsel for both companies, management staff, and others as needed. An actuarial firm, first retained three years previously, was able to support a premium rate schedule about 21% below the competition. These low rates were made possible by the absence of 5% profit and 3% to 5% agent fees, and use of investment income from reserves to discount premiums. In commercial companies this very considerable sum usually goes into general funds as profit. There remained the problem of a capital surplus required by the insurance commissioner as well as by CMIC plans for conservative management.

---

* Joseph Sadowski, M.D., chairman; Laurence B. Ahrens, M.D.; Sultan Ahamed, M.D.; W. Raymond James, M.D.; Kevin Dowling, M.D.; Dr. Jerome K. Freedman, M.D.; and James Orphanos, M.D.

W. RAYMOND JAMES, M.D., is a member of the CSMS Council and alternate delegate to the AMA. Dr. James is past president of CSMS and has been in the private practice of medicine in Essex, Connecticut since 1948.

First-year plans called for contributed surplus from each of the insureds equal to 60% of the premium. This would pose a considerable burden to insureds even though the surplus contributed would remain intact. The state insurance regulations precluded any statement about later return of contributed surplus, although most CMIC potential insureds realized that this expense would be matched by savings from lower premiums. Arrangements were made with a bank for three-year loans to interested insureds to spread the surplus payments. With this plan the sum of premiums and surplus due each of the first three years was approximately the same as competitors' premiums, but CMIC insureds would be accumulating surplus. By 17 August progress was satisfactory and the CSMS council approved the Ad Hoc Committee's recommendation to proceed.

Much time and effort was expended between 18 July and 15 October 1984, when the company became fully operational. AIS presented the Committee with an "Implementation Schedule" as well as a detailed budget covering the period from July to November. Included were over 150 items such as: planning for organization of administration and management; selecting and training office staff; locating temporary and permanent offices, equipment, and supplies; selection of banks and auditors; designing internal and investment accounting systems; preparing and distributing of solicitation packets; developing commitment forms and policies; verifying status of prospective insureds; filing extensive forms with the state insurance department with biweekly reports and meetings; marketing, including presentations to medical staffs, county associations, specialty, and county committee officers and committees; appointing reinsurance brokers and negotiating reinsurance terms and conditions (this was done through Lloyd's of London for losses over $250,000, with John Dorsett as agent for CMIC, accompanied by four board members); appointing board and committee members and organizing each of these; developing and approving by-laws; obtaining attorney general and Securities and Exchange rulings; and completing contracts between AIS, Connecticut Medical Management Incorporated (CMMI), CMIC, and CSMS.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

In the midst of all the frenetic start-up activity, on 24 August 1984, a day known ever since to the CSMS staff as "Black Friday," the executive director received two phone calls within the same hour notifying the Society that the boards of the two largest county associations, Fairfield and Hartford (representing over half of CSMS membership), had voted to endorse CNA for the coming year instead of CMIC.*[1] Stunned, even the ebullient John Dorsett feared that the project was doomed. However, the executive director, Mr. Timothy Norbeck, in consultation with the CSMS president, Dr. Joseph Sadowski, polled all the councilors by phone as to whether to proceed with the start-up of CMIC. By the end of the day the answer was given clearly to "stay the course" by a 31 to 3 vote,[1] and the team felt a strong determination to succeed. Weeks later another dramatic event, galvanizing physician leaders and staff alike, was the discovery and exposure of an uninvited CNA "plant" caught infiltrating a crucial statewide campaign strategy meeting.

Perhaps the most intriguing matter during organization and the next four years involved the role of CSMS in the management, and particularly the policy-making processes, of the new company. Originally prime movers in the "Blues" and other medically related organizations, medical societies soon found themselves excluded from policy making (the "Blues syndrome"). Extensive discussion of the role of CSMS in CMIC was discouraging, particularly in policy making or approval. Any such role by CSMS would give noninsureds a voice in determining CMIC policies and was contrary to state statutes. Thus the "Blues Syndrome" had the usual effect (ie, no voice by the Society in policy) before the company was even started!

---

*A follow-up letter from the Fairfield County Medical Association Board recommended that all relations with AIS be severed and CMIC be put on hold.

All that could be done was to include in the CMIC by-laws the requirements that all insureds must be CSMS members, and that CSMS have a limited veto over some nominations to the CMIC board. Consequently, the CMIC board has maintained a reasonable geographic and specialty equilibrium. New by-laws specify county distribution and require a nominating committee to consult the county associations involved before recommending nominees to the membership.[2] Almost all recent CSMS presidents are, or have been, members of the CMIC board of directors,* assuring linkage between CMIC and CSMS.

The original characteristics of CMIC, including by-laws, agreements with insureds, as well as company policies and regulations, were determined by CSMS and, in the start-up period, through the Ad Hoc Committee. The Society also contracted with AIS to start and temporarily manage the company and appointed the original board of directors. It was the Society's decision to have the company managed and operated by its board of directors instead of using a management company, as do some captive companies. CMIC continues to use the logo *Established in 1984 by the Connecticut State Medical Society* on letterheads and reports.

Implementation seemed to be proceeding very well. The implementation budget, totaling some 480,000 dollars, and the monies spent three years before in making the original plans, emphasized the importance of success. Failure would cost the Society over a 500,000 dollars. In early September, as sales of insurance and solicitation of contributed surplus were to begin, a completely unexpected notification was received from the insurance commissioner prohibiting any solicitation for surplus until the company held a surplus of at least one 1,000,000 dollars! CSMS was and is a very sound society, then nearly 200 years in existence, and was working with AIS, a company with 100% success in starting six similar companies. Since projected plans were conservative, it was surprising that the commissioner should suddenly impose such a seemingly impossible condition. Fortunately, the Society had been in close contact with a successful physicians' liability company in New Jersey and with their board chairman, Dr. James Todd. Mr. Dorsett and AHS had also helped organize and start that captive company. The CSMS Council then voted 19-3 to guarantee a loan for 1,000,000 dollars from the New Jersey company. The next day there was a 1,000,000 dollar surplus in the bank and CMIC started mailing solicitations for insurance and for contributed surplus! It took less than three weeks to return the 1,000,000 dollars.

There were other worrisome areas in marketing. Nearly half of the HCMA membership belonged to the Hartford Hospital staff. The joint hospital and staff insurance plan underwritten by Aetna had proven very satisfactory and not many members could be expected from that group. Also, the insurance committee in Hartford (DOMAIN), had endorsed CNA in August and withheld endorsement of CMIC until the last minute, eventually continuing endorsement of CNA as well.[3]

The leadership of the State Society from the FCMA had been active and supportive of CMIC from the first consideration of a captive company through the selection of AIS to do a feasibility study and then to organize, charter, and operate the new company. However, there was also in the association a nucleus of opposition to CMIC and particularly AIS. The FCMA was the last of the county associations to support the new company, eventually endorsing CMIC in March 1985. Response from the membership in Fairfield County, however, was good from the beginning. Reorganization of the IPA in New Haven County had resulted in new leadership supportive of CMIC.

---

*Dr. Guy Van Syckle (1975), CMIC Bd. 1984-89; Dr. Jerome K. Freedman (1978), Ad Hoc Committee, CMMI Bd. 1984-92 (Chair. 1984-86), CMIC Bd. 1988-92; Dr. Edward A. Kamens (1979), CMIC Bd. 1984-88, CMMI Bd. 1990-92; Dr. W. Raymond James (1980), Ad Hoc Committee, CMIC Bd. 1984-91, CMMI Bd. 1984-90, Chair. Claims Management Committee 1984-91; Dr. Gioacchino S. Parrella (1984), CMIC Bd. 1984-88 (retired); Dr. Daniel W. Doctor (1983), CMMI Bd. 1984-90, CMIC Bd. 1990-92; Dr. Joseph Sadowski (1984), Ad Hoc Committee, CMIC Bd. 1984-92, CMMI Bd. 1984-92, Chair. Nominating Committee 1989-92; Dr. William A. Whalen (1985), CMIC Bd. 1984-88 (deceased); Dr. James P. Orphanos (1987), Ad Hoc Committee, CMMI Bd. 1984-89 (Chair. 1986-89), CMIC Bd. 1984-90; Dr. Mehdi S. Eslami (1990), CMMI Bd. 1984-92 (Chair. 1989-92), CMIC Bd. 1984-92, Chair. Marketing Committee 1988-92, Chair. By-Laws Committee 1991-92; Dr. Sultan Ahamed (1992), Ad Hoc Committee, CMIC Bd. 1986-92, CMMI Bd. 1984-92, Chair. Finance Committee 1987-92; Dr. Theodore Zanker (Vice President 1992), CMIC Bd. 1984-92. In addition, Mr. Timothy B. Norbeck, Executive Director of CSMS, has been a member of the CMMI Bd. and Treasurer 1984-92.

Geographic variations in losses, thought to be real when AHS prepared the first premium rate schedules in 1981, proved to be illusory. Even so, the Aetna Insurance Company still offered lower premiums in the smaller counties, discontinuing only high-risk policies. The St. Paul's Insurance Company was no longer soliciting individuals, although they retained some group policies at favorable rates and with lower protection limits. CNA continued vigorous marketing but without the premium reductions used in 1981. Other than a few national plans involving only members of several subspecialties, there were no other insurance companies offering professional liability insurance in Connecticut.

Initial reaction from county and state officers and in medical staffs and other groups to CMIC presentations seemed favorable but with some confusion about the purpose of a separate management company. Emphasized, in addition to lower costs, were: better claims management with extensive peer review by insureds; payment only for well-recognized liability and strong defense of suits with doubts as to physician liability; the right to refuse settlement; improved risk management; physician education through peer review and analysis of causes of loss; and close contact between defendant physician, defense lawyers, and CMIC.

By 27 September 673 applications had been accepted and the insurance commissioner granted permission to bind policies effective 1 October. By 15 October this number had risen to 925, assuring the success of CMIC. The company was settled in new offices, the CMIC committees were operational, the implementation plans were essentially completed, and preparation of the company's "Operation Manual" was well under way.

The company had originally intended to sell primarily "occurrence" policies which provide the maximum protection for tinsureds but at slightly higher rates than "claims-made" policies. In the mid-1980s huge settlements in physicians liability, product liability, and other liability cases were a strain on reinsurance companies. There were even rumors of impending bankruptcy of Lloyd's. The decreasing availability of reinsurance and reluctance of reinsurance companies to cover occurrence policies forced CMIC to discontinue them after the first two years and market only claims-made policies (modified by coverage for claims arising after the last required premium year for retiring or disabled physicians). In the first year 10% of the reinsurance was refused by several companies that Lloyd's had expected to participate. (Reinsurance is usually distributed among many companies each covering a small share.) The difference was fortunately available through Aetna's reinsurance division. For several years reinsurance was not available for the upper limits that had been chosen (three to six million dollars). In the past few years reinsurance rates have been more favorable to CMIC. In 1992 the company again sold policies with limits of five to eight million. The company now reinsures for losses over 500,000 dollars on each individual claim. It is to be hoped that the large reserves and good record on indemnity payments and defense costs, as well as the recent decline in incidence of cases in Connecticut, have impressed reinsurance companies favorably.

The original CMIC by-laws and contracts with CMMI and AIS provided for five standing committees. These were committees of CMIC appointed by, and reporting to, CMIC but also working with CMMI and, of course, mostly with the staff. Included were: claims management, chaired by Dr. W. Raymond James; risk management, chaired by Dr. William A. Schear; underwriting and marketing, chaired by Dr. Clayton Reed; and finance (pensions added later) and executive committees both chaired by Dr. Kevin Dowling. In 1988 marketing was made a separate committee, chaired by Dr. Mehdi Eslami, with Beatrice Holt heading the new department. In 1989 a nominating committee was created, chaired by Dr. Joseph Sadowski. Some of the committees may, and do, include nondirectors approved by the board and acting partly exofficially (ie, policy and money votes must include a majority from board members for approval). The claims committee in particular has such members in order to have more extensive specialty and geographic representation. Board members and committee members

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

receive a stipend for meetings, which are usually quite lengthy, and members are paid a reasonable amount for chart reviews. There is also an arrangement for compensation to certain counties for peer review activities related to CMIC.

Most of the AIS staff had been selected and trained before the first meetings of the boards and committees, with the position of claims manager filled in June 1985. Several well qualified candidates for this position were interviewed by AIS and the committee. All unanimously supported the AIS decision to employ Mr. Gerhardt Haas, an outstanding addition to the already excellent staff. The staff and committee also prepared a claims management manual, with some later modifications at Mr. Haas's suggestion. The manual detailed the organization and procedures of the committee and the department and established the policies that were to guide the management of claims. These policies included: to serve the interests of the insureds in a fair, efficient, sensitive, and responsible manner; to defend claims aggressively in which medical and legal standards have not been breached; to act promptly to settle meritorious claims; and to retain only the most capable, experienced, and reliable defense attorneys (nearly 80 attorneys from about 20 companies have been on the board-approved list, and recently the department has also employed a full-time attorney to facilitate claims development and defense). Other sections include timeliness; settlement; reserving the role and relationships of claims representatives with the insureds; and physician review by the claims committee, county claims review committees, selected specialists, and others at the request of the defendant.

There were only two claims the first October. Thereafter the rate of claims per insured increased steadily until 1989 when the incidence of claims began a steady decrease. By October 1992 there were 3,443 files of which 2,238 were closed. Of the suits completed in court by the same date 285 were decided for the defendant physician and 123 were plaintiff decisions.[4] Note that CMIC's incidence of claims is high because of company efforts to have all problems reported early when the company can act quickly and with better results. Company policy permits claims representatives to settle the smaller cases, the claims manager and the committee settle most claims or recommend defense with an estimate of possible loss. Claims involving large amounts and questionable cases must be approved by the CMIC executive committee or the board. Since 1988 a policy of covering early costs for injured patients and family has avoided some antagonism to the defendant physician and has reduced some of the eventual costs, as well as benefiting the patient and family.

A defense attorney's manual outlines the company's expectations from defense counsel and their relationships with defendants and the claims department. The department regularly reviews attorneys' fees and has prepared a guide to reasonable fees that seem acceptable to the attorneys. Claims representatives try to attend all trials, providing the department and committee with information on defense attorney performance and contributing to the defense. Annual half-day seminars with excellent attendance by defense attorneys, CMIC board members, claims committee members, and state and county officers have been informative and very popular.

Early experience in claims management revealed a larger number of complications than expected from biopsy during colonoscopy. Chart reviews revealed no connection to specialty, diagnosis, or hospital. The risk management committee reported this to the membership, and the underwriting committee reviewed the data in relation with premium rate setting in general and for the evaluation of involved physicians. The claims committee also made an extensive study of cases related to cancer of the breast (36) with analysis of deficiencies in diagnosis and treatment. This has been distributed to the membership and also published. Other studies are underway by the risk management as well as the claims management committee.

In their society endorsed plans Aetna and CNA had included county-oriented risk management programs with expenses covered. CMIC has added many new features in a greatly expanded risk management program, best known of which have been the five self-study courses developed by the risk management committee. Successful completion of each course, requiring five to six hours, entitles a member to a 5% reduction of the basic premium for the following year. Reports on this program have been given by the committee chairman and staff at the Physician Insurers Associations of America (PIAA), and to other insurance companies and risk management meetings. A number of these organizations have arranged for use of the program. Seminars were given during the first two years for all members. Similar seminars for new members are required. Failure to participate means loss of the 25% reduction in premiums granted to members in their second year of practice. Seminars for office employees have also been available. The risk management committee and staff have worked extensively with hospital risk management and utilization review programs. Support programs for defendant physicians and their spouses, meeting in small groups with a defense attorney, a psychiatrist, a committee member, and usually a physician with prior experience, have been well received as has individual counseling. A program of follow-up of defendants and plaintiffs is expected to be beneficial to both, as well as to provide information as to why suits occur. Although CMIC believes the strong risk management program has contributed to the declining rate of claims since 1988, a similar though lesser reduction has been occurring throughout the country. Immense damages awarded in product liability suits, as well as suits against towns and even nursery schools, have led to support for tort reform, an area where physicians have stood alone in the past. Recently an impressive program on national television highlighted some unwarranted suits and immense awards, faulting the very large incomes derived from these cases by trial lawyers. It is to be hoped that jurors are being impressed with the need for reason in making awards, and legislators with the need for further tort reform.

All applications for insurance have been screened by the underwriting staff and committee using standards established in the underwriting manual. Suspected high-risk or inaccurate applications are also reviewed by the executive committee before insurance can be denied or approved. This area is closely watched by the insurance commissioner, who considers it necessary that all state residents have a source of redress if injured by a physician. This imposes limitations on how many, if any, licensed, high-risk physicians can be left with no insurance. Fortunately most physicians denied by CMIC had been insured previously by other companies. An alternative for high-risk physicians, used occasionally, has been premium surcharges.

In 1990 the marketing committee, concerned with a slower growth than expected in spite of an extensive marketing program, undertook a survey of nonmembers which confirmed that the requirement for capital contribution continued to be the leading reason given by physicians for not insuring with CMIC. The size of the company and insurance available through some subspecialty societies were prominent among other reasons given.

Although contributed surplus was a necessity in the early years, the success of the company no longer required additional surplus to maintain stability. Surplus has always been returned to retiring or disabled physicians and to those leaving the state. Further returns would reduce the surplus-to-premium ratio below the desirable range. The long period of time required to settle cases (the national average is over five-years) makes prediction of profit and loss unsure for years. It now appears there may have been a profit in several past years which will increase the surplus. Large surplus and reserves, and diminishing claims frequency, led actuarial consultants to recommend level premium rates in 1990 and 1991. Individual specialties were reduced based on individual experiences in 1992 and 1993, with the basic rates remaining unchanged. Under these conditions the marketing committee recommended that, after 1 October 1990, new members not be assessed for surplus. This decision was understandably hard to accept for many of the early supporters who had taken the largest risks. Consequently a plan was developed, and approved by the insurance commissioner and the board, giving members dividends as premium discounts in proportion to length of their membership. Eventually, if this plan can continue, the discounts will repay the early supporters all the surplus they had contributed.

During the formation of CMIC, John Dorsett, concerned over the "Blues Syndrome," suggested the formation of a second corporation, Connecticut Medical Management Incorporated (CMMI), owned by CSMS, and contracting with CMIC to manage the operation of

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

CMIC. CMMI would then contract with AIS for the formation and management of CMMI for the first three to five years. This plan seemed to increase interaction between CMIC and the Society, appealing to strong CSMS supporters, but it did not solve the "Blues Syndrome." All policy decisions would still remain with CMIC. More thought and experience by the members of the ad hoc committee, as well as AIS and CSMS attorneys, might have led to realization that the operation of an insurance company with no role in policy making was not the kind of enterprise a medical society would want to undertake.

Mistakes in the organization of the two companies precluded the success of this plan. The standing committees that were to have major roles in policy making and operation of the companies were committees of CMIC, appointed by and responsible to CMIC. At the same time the contract with CMMI, and in turn with AIS, called for much the same activities and responsibilities. The staff then was responsible not only to their employers (AIS and later CMMI), but also to the committees and CMIC. In spite of this complex arrangement, and to the credit of the excellent staff, operation of the companies by AIS was efficient with a minimum of friction, but some duplication of reports, paper work, and staff time.

The CMMI contract with AIS included reimbursement of a decreasing percentage of premiums beginning at 8.7% of the first 10,000,000 dollars of premiums earned on the basic policy, plus 7% earned on policy limits over 1,000,000 dollars, and 5.8% of additional premium income. The calculations had been based on occurrence policies, and when CMIC had to change to claims-made policies the premium income markedly decreased, forcing a renegotiation of rates to be fair to AIS. The contract for five years allowed time to have an experienced and well-trained staff, business procedures, data processing, etc, in place. Provision was also made for earlier termination in three or four years, with a lump sum settlement to repay the very considerable start-up costs assumed by AIS which they expected to make up from income in the fourth and fifth years.

On 27 February 1987 the chairmen of CMIC and CMMI, apparently motivated by a belief that AIS charges were too high, initiated talks with AIS owners in San Francisco concerning modifications of the contract.[5] They apparently gave AIS the required six months notification from CMMI of intent to terminate the contract on 1 October 1987. These actions were reported to the CMMI board at a special meeting on 21 April 1987. At the same meeting it was voted to employ a New York based firm with a local branch to make an independent study of CMIC and CMMI with a report and plan for the transition of management and operation of CMIC from AIS to CMMI.[6] Their report, dated 21 July, with a preliminary report in June, was quite positive and included analysis of future growth and suggestions for additional personnel as the company expanded and AIS functions ceased. The present staff was described as dedicated, with strength in the departmental managers and the general manager.

On 13 March, as contracted, AIS prepared a similar report, including an outline of future growth and need for additional personnel.[7] Both reports included items to be completed for the transition and a time table to guide CMMI in the process. The agreement with AIS allowed CMMI the opportunity to offer positions to all AIS staff and employees involved in the operation of CMMI before AIS would approach them. Fortunately only two staff members chose not to remain with CMMI. AIS (Denise Funk was chief of operations in all of Connecticut for AIS as well as head of the staff at CMMI for AIS) accomplished all the outlined steps including salary agreements, development of personnel policies, and contracts for fringe benefits. These were approved by the CMMI board and on 30 September the board chairman, AIS, and attorneys met and settled the remaining details necessary to complete the contract.

Two new problems had become apparent during the separation period. The first concerned liability for sales tax for services performed for CMIC by AIS and CMMI. These taxes were eventually settled for very little but the attorneys could give us no assurance that the entire 7.5% might not be assessed. The second concerned differences in pension systems between CSMS and CMMI. From the standpoint of the Internal Revenue Service both staffs were employees of CSMS. CMMI and CSMS lawyers reported considerable difficulty in that CSMS pensions were of the defined benefit type, and the CMMI plan was for a defined contribution type. A change in either plan could have been costly.

These problems, as well as the possibility of others arising from the complex relationships between CMIC, CMMI, and CSMS, were resolved in 1988 with the termination of the management contract between CMMI and CMIC and the transfer of all staff, equipment, leases, etc. to CMIC. CMMI continues to function as liaison between CMIC and CSMS, and is still responsible for all reporting to the CSMS council and House of Delegates. CMIC minutes and related materials go to each CMMI board member, and each is invited to attend CMIC board meetings. It has been suggested that CMMI is now redundant.[8] Several board members expressed the opinion that direct reports from the chairman of CMIC to the Society would strengthen the ties between the company and the CSMS. On 24 February 1992, the CMIC board approved a resolution that:

> CMIC shall provide to CSMS a grant-in-aid for the amount of $25,000 for the calendar year of 1992, which shall be used for programs or activities that have the purpose and effect of: (1) supporting and improving the quality of medical care delivered by physicians and surgeons in Connecticut; (2) reducing the frequency and/or severity of claims and suits brought against physicians and surgeons in Connecticut, and (3) educating and informing physicians about professional liability insurance.

The steady growth of CMIC required more space than was available in Wallingford. In August 1990 the company moved to new quarters in Glastonbury as part owners as well as tenants. This was in line with John Dorsett's recommendation to locate near Hartford which is a major insurance center as well as the state capital.

The Society's venture into the professional liability insurance business has been impressive, with CMIC insuring over 60% of the Connecticut market. Premiums are generally less than competitors and the company is accumulating surplus. The annual budget (cost of managing and operating the company) is below 10% of premiums (it is about 22% for competing insurance companies). By 1 October 1992 the net payments for indemnity and defense costs for the first eight years came to 33,969,917 dollars. Current reserves committed to open files are 83,706,028 dollars for the same period. The company has over 27,000,000 dollars in surplus and over 200,000,000 dollars in total assets invested.[9]

The company has an equally impressive record of physician involvement in areas that are leading to reductions in "malpractice" and patient injury. Achievements by the risk management committee and department have received national recognition, and the committee is proceeding in new areas that promise to be very productive. The highly regarded claims management department and committee have involved many physicians in case reviews and developed relationships with defense attorneys that promise a bright future. The breast cancer claims study, and others now in progress, are producing valuable information as well as educating members. These activities, as well as others mentioned in this report, illustrate advantages beyond the financial savings accruing from CSMS's foresight in founding a physician owned and operated professional liability insurance company.

Our forebears of 200 years ago would be astounded by the explosion of medical knowledge and the quality of medical care available today. They would probably be even more amazed to find that malpractice litigation, almost unknown to them, is now a constant threat to the most competent physicians performing the wonders of modern medical care to the best of their ability. Or that a major activity of their medical society would become the defense of its members from litigation and accusations by their patients over imperfect results that must inevitably occur in spite of advancing knowledge and skills, because physicians are human and cannot be perfect.

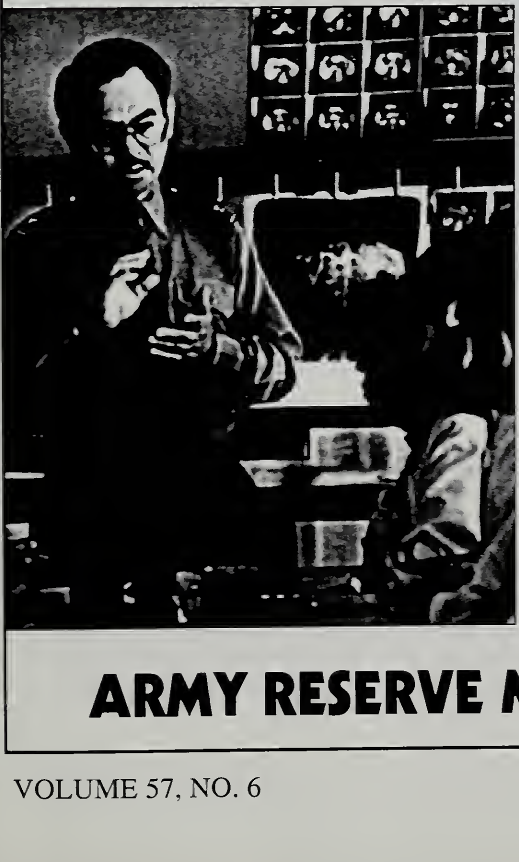
HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Perhaps our professional successors in the CSMS tricentennial year will look back at the appearance of the captive insurance companies as the beginning of effective progress in control of the many areas of medical malpractice. Surely, the extensive data now being gathered by the PIAA and others will provide a foundation for fruitful research. The burgeoning problems in health care, including the inexcusably high cost of malpractice to society, the physician, and the injured patient, we hope will convince the physician and the public that there must be better understanding of medical malpractice, its causes ,and how it can be contained.[10]

REFERENCES

1. Minutes, CSMS, 6 September 1984. Report on CMIC by CSMS president, Sect. B, and personal communication with the executive director of CSMS.
2. CMIC By-Laws approved 21 February 1990, Article III, Section 1 and 2.
3. Minutes, CSMS, 6 September 1984, Report on CMIC by CSMS president.
4. CMIC current unaudited claims statistics, 30 September 1992.
5. There is no record of studies on the comparative costs.
6. Deloitte, Haskins, and Sells.
7. See "CMMI Transition Plan," 13 March 1987, from AIS.
8. CMIC minutes 20 May 1991, page 3 "CMMI report."
9. Personal report from Emmanuel Zervos, vice president in charge of finance, October 1992.
10. The bibliography and notes are fairly complete for recorded information but not all events described in the last two articles were recorded. All such events were personally observed by the author who was a participant in most of the society's malpractice insurance activities since 1972. This included being vice president, president-elect, and president of CSMS, 1978-81; councilor 1972-86; alternate delegate to AMA 1982-86; member of the ad hoc committee in 1984; member of the board of directors of CMIC and CMMI from 1984-91; chairman of the claims management committee 1984-91; member of the executive committee of CMMI 1984-86; member of the executive committee of CMIC 1984-91; and also attending many committee meetings, negotiating sessions, and state and national conferences.

THE ARMY RESERVE OFFERS UNIQUE AND REWARDING EXPERIENCES.



As a medical officer in the Army Reserve you will be offered a variety of challenges and rewards. You will also have a unique array of advantages that will add a new dimension to your civilian career, such as:

- special training programs
- advanced casualty care
- advanced trauma life support
- flight medicine
- continuing medical education programs and conferences
- physician networking
- attractive retirement benefits
- change of pace

It could be to your advantage to find out how well the Army Reserve will treat you for a small amount of your time. An Army Reserve Medical Counselor can tell you more, call: 1(800)432-7279

Call collect (203) 624-5325

ARMY RESERVE MEDICINE. BE ALL YOU CAN BE.®

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Because One Size Doesn't Fit All...

Eli Lilly and Company can suit all your needs with the most complete line of human insulins available.

Featuring Humulin 70/30* and our latest addition to the premixed line, Humulin 50/50† —especially useful in situations in which a greater insulin response is desirable for greater glycemic control.

Humulin®
human insulin
(recombinant DNA origin)

Tailor-made options in insulin therapy

WARNING: Any change of insulin should be made cautiously and only under medical supervision.

*Humulin® 70/30 (70% human insulin isophane suspension, 30% human insulin injection [recombinant DNA origin]).

†Humulin® 50/50 (50% human insulin isophane suspension, 50% human insulin injection [recombinant DNA origin]).

Lilly

Global Excellence in Diabetes Care
Eli Lilly and Company
Indianapolis, Indiana
46285

HI-7918-B-349310 © 1993, ELI LILLY AND COMPANY

A Doctor's Child Is Born

DERRIK F. WOODBURY, M.D.

IN trying to be a New Age sensitive physician I have made it a practice to try to read most of the books written by doctors who have had illnesses and been forced into a reversal of roles. In medical school I conscientiously warmed speculums before gynecological exams. I learned that women were not girls and old people were not called "dear." Before we took our dog on a plane I even sampled his flight medication first to make sure it would be safe for him. I'm big on empathy and trying to put myself into the other person's shoes. Not holier-than-thou, mind you, but in a jockish, pizza-and-beer kind of way.

But nothing prepared me for the birth of my first child.

You see my wife is a doctor, too. She's an anesthesiologist and I'm an orthopedic surgeon. And although I delivered 20, maybe 25 babies when I was a medical student at Dartmouth—and the process hasn't changed all that much in the last few years—it seemed like a whole new world to me.

My wife Kate was working in the operating room when her waters broke. We didn't experience that mad dash to the hospital that we all read about and dread. She just went up one flight of stairs from the operating room to the delivery floor. When she got there the staff thought someone had mistakenly paged her to help with a caesarean section or to give an epidural anesthetic.

"Hi, Dr. Woodbury" they said. "Sorry, but no one's in labor right now."

Kate really did still look trim in her blue scrubs and her white lab coat. "I am," she said.

Kate calmly called me at the office and I finished seeing my patients. Obediently following instructions, I then went home to feed our dog and give him one more pep talk about the new friend we were about to bring home. I also picked up the "stuff" for the hospital stay. As one comic wag has said, you get to a certain point in your life and you can't go anywhere without your "stuff." I had just reached that point in my life.

I arrived at the hospital about 6:00 P.M. and Kate was already situated in a birthing room. The vigil began. After all the rushing around, I found very little happening. I had sort of expected everyone to be running about in a frenzy—I mean, we were about to have a baby! But that fact did not seem to have ignited the crowd. Most of the staff were watching the evening news and Kate was reading her birthing class crib notes. We settled in for the vigil.

As the night wore on Mom and Baby were doing well but labor wasn't progressing. Kate was 50% effaced—that is, the rim of her cervix had substantially thinned out—but she was stuck at two centimeters dilation. The nurses were lobbying hard for some Stadol (a synthetic narcotic analgesic) but Kate was resisting—she didn't want to feel "doped up" and miss the experience. But their arguments eventually wore her down and reluctantly she allowed the nurse to give her a small dose. I stayed out of this discussion for the most part. It seemed like a philosophical debate between people who were much more informed and experienced than myself. The medication worked though. It relaxed her and the baby descended some more.

Everyone kept telling me to get some sleep—I wasn't sure if they just wanted to get me out of the way or whether they were truly concerned for my health, but I couldn't sleep a wink. I tried to placate them and lie down, but, no way.

Kate was dozing from the effects of the Stadol and I rubbed her back and murmured support. I watched the

---

DERRIK WOODBURY, M.D., Orthopedic surgeon, Avon and Windsor. Adapted with permission from the Spring 1993 issue of *Dartmouth Medicine* magazine.

"Tonight Show," "Letterman," "Bob Costas," and the "Late Night News." They all blended together.

Leo, an anesthesiologist colleague of Kate's, came in to do the epidural. "You'll have to leave the room now," the nurse said to me.

Leo paid me the ultimate professional compliment: "No he can stay," he said lightly. "I can always use a good hand."

Unbeknownst to Leo, Kate was glaring at me while this exchange was going on. There are many good reasons for asking the father to leave the room during an epidural procedure: No one needs a father fainting, falling, and cracking his skull while the doctor is trying to manipulate a needle around the spinal canal. No matter how grizzled a veteran of the medical wars, it is not a pretty sight to see an invasive procedure performed on a loved one. And few family members would he able to be of any real assistance in a medical emergency.

Leo sat Kate upright and prepped her lower back with Betadine. He then placed the classic drape with a donut hole cut out of the middle over her lumbar-sacral region. As he penetrated Kate's skin with the needle I tried not to think about the tissue planes. But as the needle slid in deeper and deeper I couldn't stop myself. As if I were manipulating the needle I saw in my mind the space between the spinous processes. Leo angled up ever so slightly to account for their dip south. The needle then passed through the interspinous ligament. Then, deeper still. It must be at the posterior longitudinal ligament now. As the needle reached the ligamentum flavum I tried not to picture a venous plexus or arterial tributary in the epidural space. Then my mind went on red alert: "Halt! Don't break through the dura! Don't sneeze, Leo! Don't anyone bump his elbow and make him jam that needle into my wife's spinal canal!"

Only I didn't say any of that. I sucked it in as I would my stomach if I caught someone eyeing my profile and listened to my wife make small talk with her colleague. When Leo took the stylet out, inserted the catheter, and began to inject the anesthetic, I gazed coolly at the television screen. I was not a forbidding attending surgeon in this setting. I was out of my element. Ta-pocketa, ta-pocketa. The Walter Mitty of husbands. That's how I felt.

The epidural helped a great deal, but its effects were a little patchy. There were skip areas where the anesthesia didn't seem to have had an effect, and Leo thought maybe the catheter was heading down in the epidural space—towards Kate's feet—instead of following the usual course, going up towards her head.

"We could do it again," he offered. "Or just give bigger doses of lidocaine."

Neither option sounded attractive to me. Without thinking I responded to his second suggestion.

"I don't want her to have a seizure because of too much medication." I felt bad the minute I said it. Leo looked stricken. I had obviously touched upon his biggest concern as well, only his mind had generously allowed him to forget that I knew about such things, and now it came crashing back to him that his every move, his every decision was being closely analyzed and graded.

"Well, she really hasn't had that much so far," he said a tad defensively. "I think we're still in a safe zone."

Kate ended the discussion. "I'm comfortable. Why don't we just leave it as it is?" I thought at first that she was just saying that to alleviate her colleague's quandary, but in hushed tones she told me she really didn't want to be too comfortable and miss the sensation of giving birth. From all I'd seen and heard I didn't think she'd exactly sleep through the event, but one has to be supportive at these times. That was the mantra preached to all the "coaches" during our birthing class: "Be supportive." So I agreed with her.

With the aid of the epidural, the pace picked up considerably. Our obstetrician, Dr. Ken Elligers, started coming in and out of the room more frequently. He placed an internal monitor on the baby by putting a bunch of wires surrounded by a plastic catheter up Kate's vagina and clipping it to the baby's scalp. I winced. How could that not hurt?

Kate's cervix began to dilate more. Four centimeters ... six centimeters ... eight centimeters ... 100% effaced ....

The real action began around 4:30 A.M., when Kate started to push. Ken Elligers stretched and stretched her vaginal opening. The baby was in a posterior position and was having difficulty rotating. Ken tried to turn the baby. The nurse tried. The head nurse tried. It seemed as though everybody who happened to walk by the room got a chance to put their arm—up to the elbow—into my wife's most private orifice.

Ken left the room, and in a moment's notice there was a contraction and the baby's heart beat slowed precipitously. Inversely, my heart raced. Kate turned onto her side. No response. She turned on her other side. No response. Ken suddenly appeared again, through some sixth sense alarm system. Ken, the nurse, and myself helped Kate up on her hands and knees. The baby's heart rate picked up again. From then on, Kate labored with an oxygen mask strapped to her face.

Kate was pushing hard now, and as the hands of the clock passed 5:00 A.M., I could see the occiput of the baby's head with each contraction. I couldn't believe it! There was the head of my baby! Under that furry mat was

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

the brain of my child. Housed in that little area were all the thoughts and feelings of a new generation—led by 23 of my chromosomes. I was jolted back to reality as a dark feeling of gloom pervaded the room, unspoken but palpable. Then it dawned on me: We weren't making great progress and Kate was showing signs of fatigue. Around 5:30 A.M., Ken made a now-or-never gesture and went to the scrub sink. He gowned and gloved. The nurses opened the birthing pack. Glistening in the bright light was a set of ominous-looking forceps.

Ken concluded that vacuum extraction was necessary, explaining to everyone in the room and to no one in particular as he placed the big suction cup on the baby's head. Then he attached the other end of the hose to a suction apparatus and started to pull. Hard.

I barely noticed the pediatric physician assistants as they arrived. Awakened from a sound sleep, the two women were rubbing the sleep from their eyes and tried to rise to the occasion. An occasion so commonplace to them, yet vitally important to me. I'm sure they were wishing we could have held off for just one more hour. Then maybe the next shift would have gotten this one.

I was quietly appalled at the force with which Ken was pulling. I couldn't help but think: That suction cup is attached to my baby's head ... which is attached to the brain stem ... and the spinal cord ... and the neck ... etc.

Ken kept on pulling and twisting. Finally, at 5:50 A.M., he took a large syringe with a long needle and injected a local anesthetic near Kate's vagina. Then he picked up a great big pair of mayo scissors and following his pudendal block, poised them above the dorsal region of Kate's vagina. One-half of the scissors disappeared into her vagina and I turned away. Out of the corner of my eye I could see his hand contract and relax. Once. Twice. A smaller movement the third time.

Kate pushed. At about 6:00 A.M. the baby's head popped out. The umbilical cord was wrapped tightly around the baby's neck. No further progress could be made. The baby's heart rate slowed. Ken tried to get a clamp around the cord, but it was too tight between the cord and the neck. He tried again. No luck. The baby was turning blue. From my perch at Kate's right knee I had a perfect vantage point. It reminded me of so many cases I'd been involved with. I'd be assisting a surgeon during a particularly difficult maneuver in which I thought I had a better angle to accomplish the task. Here, I was sure I could see an opening between the neck and the cord. But I stifled my offer of assistance and my almost involuntary desire to reach for the scissors. Ken tried again—and got it! He cut the cord. Kate pushed and Ken pulled. The baby didn't budge. Didn't "pop" out like I thought it would. Just stayed there. Blue.

The shoulder was stuck.

Kate was pooped. She had leaned down and seen the head and thought it was over. But the baby couldn't breathe yet and the umbilical cord was no longer supplying oxygen.

I bent down close to Kate and implored her to push at the next contraction. She looked at me quizzically because she really thought her work was done; she was a little dazed and approaching that state of mind where one doesn't care anymore. But something in my eyes and the tone of my voice touched her and made her reach down for one last, gargantuan effort to push. Her sleepy, fatigued brain responded and sent waves of pure willpower to her contracting uterus. Ken pulled and worked his left hand between the baby's neck and Kate's vagina. And ...

The right shoulder popped out.

For two or three long seconds the baby was frozen in that position. Then Ken rotated the whole little body, and the left shoulder popped out. Finally out came the rest of the tiny form ... motionless ... blue ... and oh, so tiny.

The pediatricians engulfed the child, and I said to it in a whisper, "Breathe ... Breathe."

"It's a beautiful boy," the maternity nurse said. "Born at 6:07 A.M."

I hadn't yet been able to determine the baby's sex, because as he was being born the twisting motion put his back towards me. They whisked his blue body to the resuscitation crib and I saw him twitch, startle, then give a tiny little cry. So faint.

I let the pediatricians do their thing and turned back to my baby's mother. Tears welled in my eyes as I congratulated her. I was overwhelmed at the magnificence of the job she had done. She had never really complained. Didn't let fatigue conquer her. She'd tried so hard. I was so lucky to have married such a person. I was so proud of my wife. I was overcome with love for this wonderful woman. That was my first emotion.

The eternity of that first minute had passed, and the pediatric physician assistant working on my baby caught my eye and said, "He's doing fine."

I stayed where I was, continuing to comfort Kate. When the flurry of activity had died down, I went over to the isolette. There he was, a little cloth cap covering his cone-shaped head. His eyes were wide open and he wasn't crying, but was breathing ever so rapidly, taking small, shallow breaths.

"What's his name?" the maternity nurse asked.

"I don't know," I replied.

I was hardly paying attention to what was going on around me. I was examining my child. His eyes seemed alert. There was no cleft lip or palate. He had five fingers on each hand. He moved all his arms and legs. He had five toes on each foot. I turned him over and fingered his spine.

It was straight. No spina bifida. I checked his hips. They weren't dislocated. Then, finally, I turned him over again and looked at his groin. He was a boy.

"What's his name?" the nurse asked again. This time the question was directed to my wife.

"What do you think, honey?" she called over to me at the isolette. I turned to her and tried not to watch Ken as he repaired his episiotomy. The huge chromic catgut needle paused in midair as he tightened the running suture. The one big operating room light reflected off the needle as he swooped back down with a flick of his wrist.

"How about Carson? Carson Fairfield?" Kate said tentatively.

"I don't know," I replied. The placenta sat in a specimen pan next to Ken, looking for all the world like a big red pancake. It appeared healthy. No calcifications, no shriveling. Not like a smoker's placenta.

"What's his name?" the nurse asked again. There was paperwork to be completed.

"Carson Fairfield Woodbury," I said authoritatively. The words came out of my mouth with much more finality than I felt. This would be his handle for life. Would it be right for him? Was I sure I liked it?

"Would you like to hold him?" the pediatrician asked. He—Carson, that is—was all bundled up now. He was securely swathed in a blanket. I took the little package gingerly. He wasn't crying at all. He was alert and looking all around him.

"He looks like you," she said.

I thanked her while thinking to myself, "I must look like Yoda."

But he looked beautiful to me.

All my years of medical training and all the nights I'd spent in hospitals had not diminished for me the miracle of this night, the miracle of the birth process that my wife and I now shared with millions of others. We were no longer just doctors. We were now parents.

I brought my son to his mother and placed him in her arms. Then I gave them both a kiss. I put my left arm around Kate and my right arm around little Carson.

I put my arms around my family.

GRATEFUL MED

$29.95

SOFTWARE
PASSWORD
$40. CREDIT

AVLINE
CANCERLIT
CATLINE(books/jrns)
CHEMLINE
DIRLINE(organizations)
HEALTH(non-clinical)
MEDLINE(biomedical)
PDQ(cancer)
SDILINE(current-month)
TOXLINE
TOXLIT
TOXNET

FREE INSTRUCTIONAL WORKSHOPS

NEW FEATURE :
USERS CAN PLACE AN ONLINE ORDER TO RECEIVE FULL-TEXT ARTICLES FROM PARTICIPATING HOSPITAL OR ACADEMIC LIBRARIES

For futher information contact:
National Network of Libraries of Medicine
New England, Region 8
University of Connecticut Health Center Library
263 Farmington Avenue
Farmington, CT 06030-5370
TEL: 800 338 RMLS or 203 679 4500

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Home to Carthage

RUSSELL V. FULDNER, M.D.

> She with whom I had lived so long was torn from my side as a hindrance to my forthcoming marriage. My heart which had held her very dear was broken and wounded and shed blood. She went back to Africa, swearing that she would never know another man, and left with me the natural son I had had of her.
>
> —*Confessions of St. Augustine*, Book Six, XV
> Translated by F.J. Sheed, 1943.

Yonder land to the east, the sailors say, is Sicilia,
The wind following since we left Ostia
We're to sight Carthage soon.
I should know the very spot on shore where Monica,
Searching for her son, was greeted with empty waves:
He told the tale so often, as we bedded down,
It surely troubled him, for all his gift of making wrong seem right.

Rome was a glory —
The circuses and games, angry beasts and gold spangled chariots, the trumpets and drums,
And best of all, the proud march of the Legion!

Monica finished it:
A bride on the way, or so she hoped, Christian to make him Christian—
He said in parting there could be none like me,
None like me, no, not ever.
He meant until I left.

For the boy I wanted so to stay,
I knew his every need and wish
And comforted him against the dark—
The joy we shared, the two of us, in his beginning steps!
May strangers bear with him tenderly,
My beautiful son.

Still, it's better Rome than Carthage,
Rome is the world.
His father will surely school him, though out of sight of Monica and the rest,
A Legion school, yes, that would be the place,
From there the world, soldier of the Legion!
Captain he'll be, perhaps tribune, then who knows?
Hail Emperor!
Hail my beautiful son!

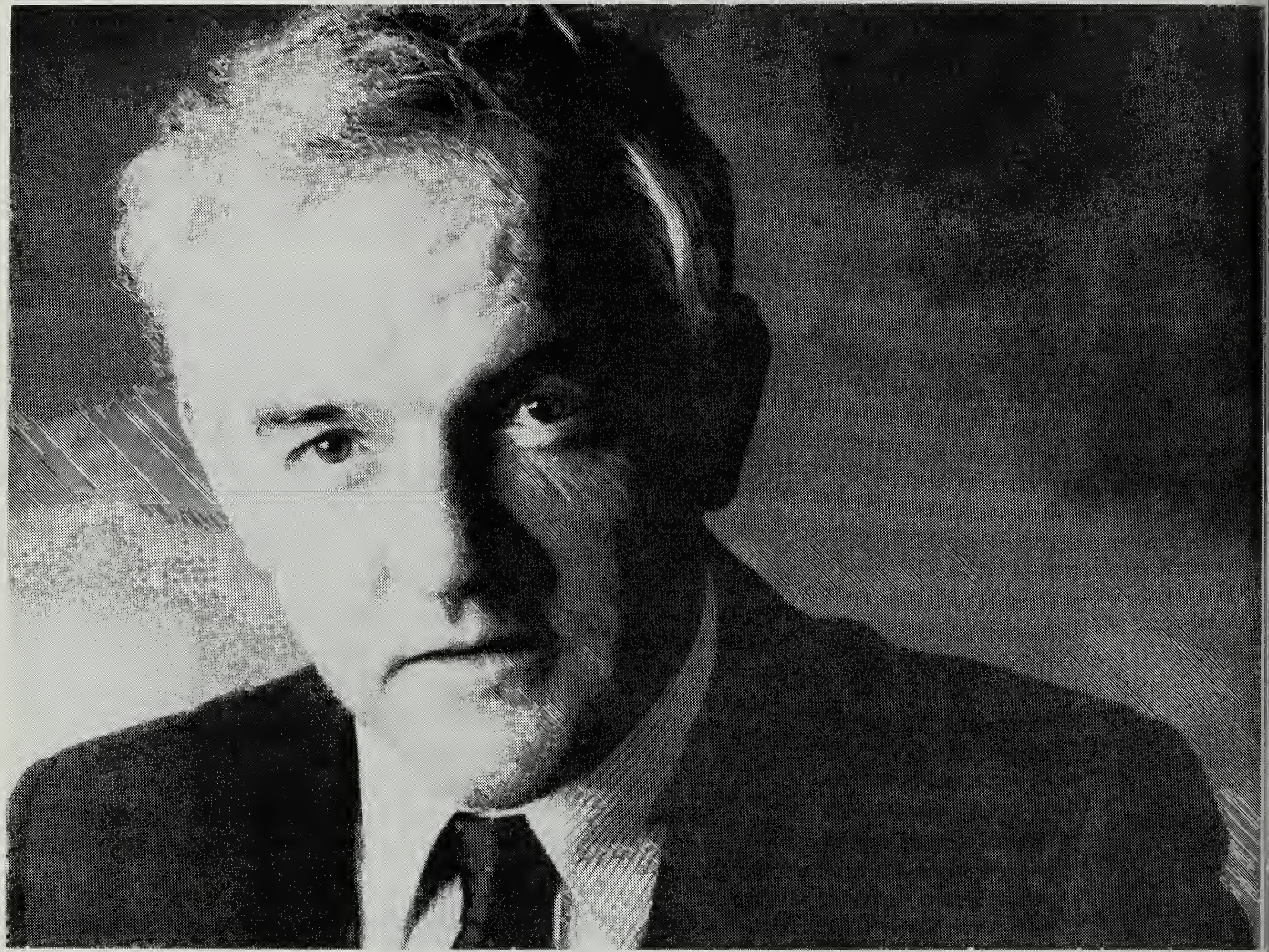
Carthage in sight and my mind utterly changed.
Freed by my son
I've decided not to plunge into the sea.

R.V. Fuldner, M.D.

RUSSEL V. FULDNER, M.D., Orthopaedic surgeon, Hamden.

“Filling out insurance forms gives my patients a pain in the neck.”

—Dr. Patrick J. Carolan, Orthopedics, Bridgeport, CT



“Good medical care was once as easy as seeing your doctor. Complications set in. Forms to fill out. Deductibles. Hidden costs. Partial coverage. Non-medical intrusions into the patient-physician relationship.

“Doctors decided to do something about it. Through our State Medical Society, we first created **CSMS-IPA,** Connecticut's only *statewide* IPA. Then we launched **M.D. Health Plan,** based on a simple principle: The best medicine for *patients* is practiced by *physicians*— not insurance companies, not benefit managers, not cost analysts.

“It worked! Now the fastest growing health plan in Connecticut and the choice of over 68,000 patients, our company is 100% owned and 100% directed by practicing physicians. Why not?”

Patrick Carolan, M.D.,

M.D. HEALTH PLAN

6 DEVINE STREET • NORTH HAVEN, CT 06473 • (203) 776-5759 • 1-800-345-9272

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

50 Years Ago

From *The Connecticut State Medical Journal*

June 1943

The International Control of Disease After the War

ALICE HAMILTON, M.D., Hadlyme

Presented at the Sesquicentennial Celebration of the Connecticut State Medical Society, June 3, 1942.

MY field has never been public health in the wider sense only a small section of it, industrial hygiene, therefore if I should attempt to discuss the future course of preventive medicine,—what should be and probably will be the developments in that field after the war—I should not only enter a very controversial field but one concerning which I have no right to speak authoritatively. The only aspect of public health aside from the industrial with which I have more than ordinary familiarity is the international control of disease, because for six years, from 1924 to 1930, I was one of the American members of the Health Committee of the League of Nations. That means that I joined the Committee when the war devastated countries were still facing epidemics which they could not control and I remained on it long enough to see those primitive conditions brought under control and a program of preventive medicine developed. That is why I feel I have a right to discuss the international control of disease which must be undertaken when this war is over, no matter what the military outcome may be, and to discuss it not as an imaginary ideal but as a program which once was carried out with brilliant success, and may be again.

For many years after 1924 whenever I mentioned the League of Nations I did so apologetically, for I knew that the great majority of my hearers had been so skillfully conditioned against it that as soon as they heard the words their minds closed with a snap and it was a hopeless task to try to force them open. So I always began by saving that it was only the work of the Health Committee that I meant to discuss and always I would find that in a short timethey were listening eagerly to a story which should have been familiar to all Americans but which was obscured by the black mantle of anti-Wilson hatred, and of resolute isolationism and xenophobia which descended on us after the war. I think everyone agrees that one of the most striking results of the second world war is the change in our attitude toward narrow nationalism and the general acceptance of the fact that after this war some kind of international organization must emerge. The first task that will face such an organization will surely be the control of epidemics and the restoration of public health service, and that is why I wish to describe the way this work was carried on between the two great wars.

The Covenant of the League had made provision for an international commission of medical experts which the Council of the League was to appoint, for they knew well that of the pressing problems facing Europe in 1919 none was more urgent than that presented by the epidemics which were sweeping over from the east. Cholera, typhus and relapsing fever had invaded Russia and were threatening that vague line which separated Poland and the Baltic states from Russia, and the strict sanitary cordon which Germany had maintained during the War had broken down. Across the boundaries everywhere, returning soldiers and refugees were pouring, most of them diseased and half starved, and there was an appalling dearth of doctors and nurses, many having died of these diseases, there were no medical supplies, no disinfectants, no soap even. Early in 1919 the Red Cross had tried to cope with the situation and had sent doctors and nurses (many of them American) into Poland, but the task was too great for a voluntary body to undertake and in 1920 the Council of the League hastily appointed an Emergency Epidemic Commission, also with American members, which did brilliant work for the two following years. One of their most remarkable accomplishments was the staging in Warsaw, in the early spring of 1922,

From the *Connecticut State Medical Journal*, June 1943.

of a Health Conference which was participated in by 25 European countries, by the city of Danzig, the Ukraine, Japan, and Turkey. Those two bitter enemies, Poland and Russia, were there; so were the new Baltic States, and the new "succession states," the defeated Central Powers and the victorious Allies, and while the statesmen of these countries could not even agree upon boundaries, they did agree to send their exchanged prisoners and repatriated refugees through jointly controlled quarantine stations.

As I read the official report of the Warsaw Conference I was struck by the tone of courtesy and respect in all the dealings with that pariah among the nations, Soviet Russia, and this at a time when most of Europe was eager to force her to submission by war or by starvation. Only the medical profession recognized the fact that disease is no respecter of nationality nor of victory nor defeat.

The Epidemic Commission also went to Greece to help in the terrible situation which developed there in the Fall of 1922, when more than a million refugees from Asia Minor, fleeing before the Turkish army, poured into Greece, bringing with them cholera, typhoid fever and smallpox. There were American physicians helping in this work also, and indeed Americans as individuals have cooperated eagerly in all the health work of the League, even when our government was holding itself completely aloof.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

By 1924, when I received my appointment, these acute situations had been brought under control and when I went to Geneva for the Autumn meeting it was to find a new set of problems waiting to be handled. The members of the Committee were then 20 in number (this varied from time to time) most of them from European countries, two, an Englishman and a Frenchman, from Africa, one each from Japan and South America, two from the United States. Surgeon General Cumming, then head of our Public Health Service, was one of the vice-presidents, and I was the second American on the Committee and the only woman. It seems that at the time of the organization of the Committee some good feminists persuaded the Council to provide for the appointment of at least one woman and, since they were then still hopeful of our joining the League, they turned to the United States and chose Dr. S. Josephine Baker, the child specialist. But Dr. Baker was unable to accept the appointment, and since they had no member from the field of industrial medicine they passed the invitation on to me for two three-year periods.

I went to Geneva full of eagerness to see the League and to meet the group of eminent experts from all over the world, but I did not look for anything exciting in the meetings where T should probably have to hear reports, largely statistical, sent in by health inspectors, with all the vividness and human interest carefully deleted. But from the very first meeting I found myself in for a very interesting experience. We met in a long room in the old Palais des Nations, sitting around three tables which were arranged as three sides of a square. Our presiding officer was a Dane, Dr. Thor Madsen, gentle, aloof, judicious, liked by everyone. The meetings were informal, Dr. Madsen announced the agenda, the men to whom an inquiry had been assigned reported on it and discussion was free.

There was absolutely no touch of narrow nationalism, perhaps because these men did not represent their governments (of course Dr. Cumming and I could not have, nor could the German member), they had been chosen by the Council because they were experts in some field of medicine, not because they were French en or British. one of the most respected members was a German, old Dr. Nocht, head of the Hamburg Institute of Tropical Medicine.

The first meeting was given over to a Malaria Subcommittee which had just finished a journey through the most heavily infested areas of southern and southeastern Europe. I was astonished to learn that, the epidemic diseases having been brought under control, malaria had become the most serious post-war problem in Europe. Of course I had read of its prevalence in the Campagna and the Mareemma, in Corsica and Sardinia, but this report covered Serbia, Croatia, Dalmatia, and Albania, Macedonia, Thrace and Greece, Bulgaria and Russia. It seems that malaria was always endemic in those countries but the war had caused an enormous increase, partly because anti-larval measures had stopped, doctors, nurses and quinine were lacking, but also because the forced migration had brought people with no immunity into infested regions and people with infection into regions hitherto free from malaria. And of course under-nourishment, wretched housing and exhausting suspense and grief added their share. An American physician told me that in Russia he had seen fulminating forms of malaria which at first he had diagnosed as encephalitis.

The measures advocated by the Malaria Commission, with Dr. Nocht at its head, were those which were considered most practicable in poverty stricken countries, which could not possibly undertake large-scale drainage and anti-larval measures. Robert Koch had shown the Italians that under such circumstances cinchonization of the population was the cheapest and simplest method, quinine for the malarial and also as a prophylactic for all those exposed. Even this is costly and there was not enough quinine to fill the need, so the Health Committee had inaugurated a study of the mixed alkaloids of cinchona bark, testing them in hospitals in Italy, Rumania, Spain and Jugoslavia.

During their journey through the malarial countries the Commission was struck with the scarcity of doctors with skill in diagnosing and treating malaria and therefore urged the Health Committee to offer anti-malaria courses for the training of experts. Here as in many other instances the Rockefeller Foundation came to the rescue and by means of fellowships made it possible for physicians from the backward lands to attend such courses in Hamburg, Liverpool, Rome and Paris. More than two hundred and fifty attended these schools.

I have gone into such detail concerning malaria because the work done in that field is typical, but I must mention briefly the second subject which chiefly engaged our attention in 1924. The Mandates Commission of the League had demanded from the three great African powers, Britain, France and Belgium, an accounting of their administration of public health in the mandated areas, the former German colonies, especially with regard to tuberculosis and sleeping sickness. Those countries appealed to the Health Committee which sent a commission of French, English and Belgian physicians to Equatorial Africa to make a survey, and report. The results of this survey were read to us. It was a tragic story to which we listened, a story of tuberculosis introduced into Africa by Europeans, Syrians and East Indians, meeting a population which has no inherited immunity, and spreading far and wide, helped on by what the report called the "advance of civilization, that is, the use of strong alcoholic drink, the increase of poverty, the change from healthful ways of life to unhealthful, and the increase of moral and physical degeneration."

As for sleeping sickness, that is a disease native to Africa but white invasion has led to a great increase. Formerly it was found only in certain districts, now with the opening up of the country through roads and the recruiting of natives for the armies or for labor, it has invaded area after area. In the control of both these plagues it was found that the regions formerly ruled by Germany were far better protected by sanitary measures than any of the others. The report ended with this significant comment "If we should put on opposite sides of the ledger the benefits and the evils which have come to Equatorial Africa as a result of the entrance of white civilization we should be obliged to say that the evil overbalances the good."

But, four years later, at the 1928 meeting we heard a very heartening tale of the measures which had been put into effect by the three ruling nations and we gained a vivid idea of the heroic work of the physicians engaged in carrying it out. Everyone knows about the brilliant anti-silicosis campaign in the mines of the Rand which used to be hotbeds of silico-tuberculosis. In the control of sleeping sickness the health authorities were greatly helped by the development of tryparsamide by the scientists of the Rockefeller Institute. Dr. Louise Pearce and Dr. W.H. Brown took it to Africa and started its use there.

Through all the discussions, especially of plans for future activities the Health Committee was faced with the difficulty of finding physicians and sanitary engineers adequately trained for public health work. Here again the Rockefeller Foundation stepped in and furnished travelling fellowships for men and women who were to be selected by their governments and sent to whatever country offered the best instruction in the particular field to he studied, the students pledging themselves to return home and enter the public health service. I had a number of these men and women in my classes at Harvard and it was a pleasure to teach them, for they were so eager to learn.

In addition to the program for the training of public health experts, that 1924 meeting saw the inception of a great scheme to obtain and broadcast all information concerning the outbreak of epidemics. Never had it been possible to make governments exchange this sort of information, on the contrary, every effort had always been made to conceal an outbreak of cholera, plague, typhus, small pox. Those of my generation will remember the time when California refused to recognize an epidemic of bubonic plague, lest tourist and real estate interests suffer, so that Washington had to send F. G. Novy, Lewellys Barker and Simon Flexner out to the coast with authority to deal with the situation. That had been the way all the countries had acted but, suddenly, perhaps because of the terrible experiences during the war, that form of isolationism broke down and the governments of the Asiatic countries as well as the European and the United States for the Philippines and the Canal Zone, agreed to exchange as promptly as possible all information concerning the five epidemic diseases, cholera, typhus, small-pox, bubonic plague and yellow fever. In later years others were added, encephalitis, meningitis, infantile paralysis, influenza. Singapore was selected as the centre for these reports which came from Vladivostok on the north to the Southern Archipelago, and from Suez on the east to the Canal Zone, and were transmitted from Singapore by wireless to public health stations everywhere. In this way, an outbreak of cholera or plague in a remote part of China or India could be made known all over the world in a few hours. The monthly, printed reports, used to come to me regularly. It is tragic to know that all that service is now at an end.

At my second and third visits, I found the Health Committee swamped with pleas for assistance. No longer did we have to approach governments tactfully and suggest that perhaps some expert advice might be helpful, the governments themselves were pleading for it.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

They asked us to look into their appallingly high infant mortality rate, and incidentally to tell them how to collect vital statistics; they told us confidentially that their budget for malaria control was small and that the big landholders wanted to spend it on large schemes of drainage which would benefit them and leave nothing for quinine for the poor; or that the government had decided to ignore malaria and spend the money on irrigation. We were asked to back up the health authorities with the prestige of the League and in both those instances this was done successfully. A request came from South America for the appointment of a Leprosy Commission, to determine the best method of control and to study the action of chaulmoogra oil which so often proved disappointingly ineffective. Brazil, it seems, has a serious leprosy problem, an aftermath of the slave trade, it is said. In certain regions there is one case to a thousand inhabitants; in a single region it is one to five hundred. Argentina, Venezuela and Colombia also have many cases. This request was backed by the oriental countries.

Then there were pleas for the standardization of therapeutic sera and vaccines, and for a commission to test and pronounce on the value of the various diagnostic tests for syphilitic infection. The Pacific powers, France and Britain, asked advice on how to stop the depopulation of the Pacific Islands where white invasion had been followed by a rapid decline in the native population. This request revealed a tragic picture of what O'Brien called "White Shadows in the Orient," the introduction of new diseases to a non-immune people, the forced deportation of men to work far from home, leaving the farms and fishing grounds to the women, which meant that canned food bought from the traders took the place of fresh food; the inevitable change in living habits, in short all the blessings of civilization imposed suddenly on primitive people.

Another, more urgent request, was sent to us in 1928 by Greece which was in the grip of an epidemic of dengue, so severe that in September of that year—we met in October—no less than 86% of the population of Athens and the Piraeus were infected, the infection being transmitted by mosquitoes and biting sand flies. When the Greek delegate came to Geneva in October he reported no less than 850,900 cases with 1,378 deaths. Greece had no public health service, and no trained personnel to man such a service. The Health Committee promptly appointed a Commission, with an Australian, a Croatian and two Americans, Haven Emerson and Allan McLaughlin. These men organized a public health service for Greece and arranged for the training of personnel in schools abroad, the work being carried on temporarily by imported experts, who would also organize a School of Hygiene, this last as well as the other activities to be turned over to the Greek students when they returned from their training abroad.

When my second term expired in 1930 I had heard reports from all these commissions, most of them very heartening, and I had seen the beginning of an interesting, study of infant mortality, the first report of which reached me later.

The Health Committee had long wished to make such a study, to discover which country had the lowest mortality rate and then to adopt as standard the infant welfare measures used in that country. But at the outset it was found that one could not compare the death rates of countries, because of the difference in their systems of vital statistics. Finally it was decided to adopt the method originated by our Children's Bureau which resulted in those very valuable infant mortality reports from cities and towns and urban regions in this country, namely to choose typical regions and make in these a detailed study of the circumstances attending every baby's death during the preceding year. This means inquiry into the family's standard of living, the fathers' earnings, the mother's work, whether in or outside the home, breast or bottle feeding, whether the child was the first or twelfth, and so on.

The countries selected were England, Norway, Holland, France, Germany, and Austria, city and country and small-town areas. The reports are extraordinarily interesting reading, bringing in all sorts of intimate details of the life of these people. The lowest death rates were found in England, Holland and Norway, with Norway standing at the head, the highest were in France, Germany and Austria. Poor Austria came out at the foot both in city—Vienna—and rural districts, in both of which there is great poverty, women's work is excessive during pregnancy and immediately after, breast-feeding is rare and artificial feeding is inadequate, and there is a high percentage of illegitimate births.

Poverty emerged as the most important factor in all these countries but a close second was bottle feeding. Breast-feeding proved to have much more influence on the death rate than had so-called infant welfare work. At the discussion in committee Dr. Velghe, of Belgium, said that during the German occupation the infant death rate fell in Brussels, in spite of the hardships of the people, simply because the women could not get work and had to suckle their babies; and Dr. Chodzko, of Poland, remarked that the same thing was noted in Warsaw under the German occupation.

In France there is a far higher infant death rate in Normandy than in the wine producing region in the South, although the Norman peasants are wealthier than the Provencals. But they are hard working and avaricious, the women will not suckle their babies because it interferes with their work in the fields. The Southerners are easy-going and there breast-feeding is universal.

The League of Nations was in principle an international organization devoted to the welfare of all, but nationalism is deep rooted and "sacred egoism" is to most "patriots" the first duty, fairness to others takes second place. The Palais des Nations was full of clashing national ambitions, of diplomatic maneuvering for position, of thinly disguised selfishness, but when I would leave the gossip filled corridors and enter the great room where the Health Committee held its sessions I would breathe at once a different air. For here nobody represented his government, all had been chosen on the basis of their scientific standing and all were working for the control of disease no matter where. Countries were rated only according to their need for help or their ability to give help. Those nations which had received help understood its importance and when, in 1928, England under Sir Austen Chamberlain, began to sabotage the League and attempted to cut the budget of the Health Committee, a storm of protest arose, especially from the countries of South America and the Far East. Those delegates said that the only activity of the League their people knew anything about was the health work. The budget was saved.

The Health Committee still exists though it has not met since the Fall of 1939. There are still some four or five members of the Secretariat left in Geneva and epidemiological reports were still sent from Singapore to Geneva and from Geneva to the United States up to December 7, 1941. Then that last international service ceased. But if ever an association of nations emerges after this war, I am sure that it will recognize the great service of the Health Committee of the League and will bring together again a similar group of experts to deal with the international health problems left by the second World War as they dealt with those of the first.

Problems of International Health

At the request of the Journal Prof. C.E.A. Winslow of Yale University offers the following editorial comment on the paper by Dr. Hamilton appearing in this issue.

As one who later had the privilege of serving with the Health Committee of the League of Nations, I can particularly commend Dr. Alice Hamilton's article on "The International Control of Disease After the War," in which she discusses some of the work done at Geneva.

The Health Section rendered invaluable service in the international standardization of sera and drugs; in special studies on cancer control, on malaria, on nutrition and housing; in the collection and dissemination of information in regard to the prevalence of organic disease; in intensive surveys and the development of special programs for nutrition in while, malaria control in Greece, control of sleeping sickness in Africa and the like; and finally by provision for the study by public health experts from various countries of the methods of procedure used in other lands.

Since Dr. Hamilton's article was written, there remains only one member of the Health Secretariat at Geneva. Much of the work it did, however, still goes on. The entire movement for the study of the hygiene of housing in the United States was initiated at the direct request of the Housing Commission of the Health Section. At the moment plans for post war health and medical service are being developed by the Leith-Ross Committee in London, and by the Office of Foreign Relief (under Governor Lehman) in this country. It is of interest to note that Dr. Gautier, the Director of the Health Section of the League, has recently been called from London to Washington for consultation in regard to this program; and it is hoped that within a very few months the whole problem of relief and rehabilitation—including health as one of its major aspects—will be turned over to a United Nations organization.

No one can tell how far the old League of Nations will be integrated with the international planning of the future; but it is clear that if it is not utilized, essentially the same machinery must be set up under another name. We may be well satisfied if whatever machinery is set up operates as well as the Health Section of the League did in the past.

From the *Connecticut State Medical Journal*, June 1943.

INQUIRE TODAY.

FOUNDED IN 1792

ENDORSED BY
THE CONNECTICUT STATE MEDICAL SOCIETY
—OFFERING—

- Hospital, Surgical and Major Medical Insurance
- Term Life Insurance
- Employee Benefit Package

—FEATURING—

- OFFICE OVERHEAD AND DISABILITY INSURANCE PACKAGE

  [Rates based on a 30 day waiting period for ages 30-39]

  $1500.00 weekly disability indemnity
  semi-annual premium is $528.15*

  $20,000.00 monthly overhead indemnity
  semi-annual premium is $561.00

* Optional Residual and COLA benefits available

ayr

AYR INSURANCE AGENCY

105 SANFORD STREET HAMDEN, CT 06514 288-4302

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

HOWARD J. WETSTONE, M.D.
President

HOWARD J. WETSTONE, M.D.

BORN:	27 April 1926 Hartford	
EDUCATION:	Wesleyan University, Middletown, B.A. with Honors, High Distinction-Biology	1946
	Tufts College Medical School, Boston, Mass., M.D.	1951
	New England Center Hospital, Boston, Mass., Internal Medicine	1951-1953
	Hartford Hospital, Hartford, Internal Medicine	1953-1955
MILITARY:	U.S. Army Air Force	1946-1947
CERTIFICATIONS:	National Board of Medical Examiners	1952
	American Board of Internal Medicine	1957
	Recertified	1977
SOCIETIES:	Fellow, American College of Physicians	1962
	Hartford County Medical Association	1955-
	Board of Directors	1978
	Legislative Committee—Chairman	1978-1984
	Bylaws Committee—Chairman	1986-
	Vice-President	1984-1985
	President	1985-1986
	Councilor	1987-1991
	Distinguished Service Award	
	Connecticut State Medical Society	1955-
	Allied Medical Services Committee—Chairman	1974-1979
	Emergency Medical Services Committee	1979-1984
	Statewide Medical Planning Committee	1979-1988
	Editorial Committee, Connecticut Medicine	1979-1983
	Legislation Committee	1978-1993
	Chairman	1989-1993
	Vice-President	1991-1992
	President Elect	1992-1993
HOSPITAL:	Hartford Hospital-Senior Attending in Medicine	
ACADEMIC:	University of Connecticut, School of Medicine: Associate Professor, Community Medicine and Health Care; Medicine	
WORK:	Private Practice-Internal Medicine, Hartford	1955-1958
	Hartford Hospital	
	Director Research, Dept of Medicine	1958-1965
	Director, Private In-Patient Teaching Services	1965-1970
	Director, Medical Out-Patient Services	1970-1972
	Director, Out-Patient Services	1972-1973
	Director, Ambulatory Services (ER,OPD)	1973-1984
	Vice-President, Corporate Medical Affairs	1984-1987
	Connecticut Health Systems	
	Vice-President, Medical Affairs	1987-1992
	MedSpan	
	Medical Director	1989-
OTHER:	Bloomfield Board of Education	1955-1969
	Chairman	1961-1969
	Connecticut Association of Boards of Education	1955-1969
	President	1961-1963
	Capitol Region Education Council: Chairman	1965-1966
	Connecticut Public Broadcasting	
	Board of Directors	1962-1991
	Chairman	1970-1974
HONORS:	Connecticut Hospital Association—Honorary Member	1981
	A.E. Hertzler Knox EMS Award	1988
	Robert U. Massey Award—Capital Area Health—Consortium	1992

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

The New President's Message

Ninety-one years ago in an address to the Canadian Medical Association William Osler described the "four great features" of our profession.

The first is our "noble ancestry," a product of the Greek intellect. It was the Hippocratic school that emancipated medicine from "the shackles" of priestcraft and caste, that conceived of medicine as an art based on accurate observation, that enunciated the "high moral ideals" of the Hippocratic oath and that saw medicine as a profession of "cultivated" people.

The second feature is our "remarkable solidarity" or universality. Osler said "the profession in truth is a sort of guild or brotherhood" (personhood would be a better word in 1993) "any member of which can take up his calling in any part of the world and find brothers whose methods and whose aims and ways are identical with his own." CSMS is, indeed, based on the model of a "hanse" or professional guild whose purpose is to guarantee the standards we deliver to our patients and to enhance our profession in general.

The third feature is the "progressive character" of medicine which advances as science advances. A science practiced with art or an art with a scientific basis, medicine was, is, and will always be one of the most exciting and challenging professions of all.

The fourth feature is the "singular beneficence" of most of medicine's practitioners. Even in today's climate of skepticism and uncertainty, it is the rare physician who does not extend services to those who need them irrespective of the potential financial reward.

The conspiratorial nature of some of our health care planners, as they pursue their strong desire for socially engineering the health care system is worrisome. Their trust in intuition rather than economically or scientifically proven facts is alarming. Their desire to exclude providers from the planning process has an aura of anti-intellectualism about it. Their view of the providers as enemies of the people is consistent with their characterization of any financially successful citizen or businesses as enemies. These modern counterparts of 19th century populists will surely test our ability to adhere to Osler's model.

What then should be our position in 1993? Simply stated all Americans should be covered by basic health insurance and the nation's physicians should accept the responsibility and make the professional commitment to do our share in containing costs.

I testified in March before the Public Health Committee of the Connecticut State Legislature that CSMS is committed to:

1. Perform professional review of excessive medical fees when antitrust laws are changed to make it legal to do so.
2. Submit to utilization review, under proper medical supervision and accountability.
3. Adopt and implement practice guidelines which can be implemented with tort reform.
4. Use outcome studies and cost effectiveness analyses to review our treatment policies.
5. Work cooperatively with all payors to reduce the administrative burdens and costs of health care.

We believe all of us should assume some level of responsibility for our own health and some accountability for the health care we consume. We continue to stress preventive health care, patient education and healthy life-styles for all patients. I urged our legislature to pass helmet laws, higher excise taxes on tobacco and alcohol, universal immunization, gun control, strict enforcement of speeding laws and severe penalties for drunken driving. If all of these were adopted and enforced health care costs would be as much as 30% lower and quality of life would be improved. Even partial legislative success would make a significant reduction in health care costs. Surprisingly, many of our legislators would rather ration or tax all citizens rather than constrain the economically wasteful health behavior of some.

In the spirit of Osler, we will work with all interested parties in achieving an affordable improvement in the health care of our citizens. We will seek cost-effective strategies with an excellent quality of life outcome. The wasteful among us will be reminded of the more prudent behavior of their peers. The small number of greedy will be reminded that the excess money they take from the system not only is costly to society but more and more comes from the pockets of their colleagues. The physicians among us with less than optimal patient outcomes will be reeducated. Those unfortunate enough to be impaired will be rehabilitated, if possible, but in any case removed from patient contact. This I pledge to you, our members, upon assuming the leadership of this 201-year-old society.

Howard J. Wetstone, M.D.
President

Inaugural Address—House of Delegates Meeting, 12 May 1993

LAW, MEDICINE, AND PUBLIC POLICY

Reexamining Liability for Acts that Lead to Death

JOSEPH M. HEALEY, J.D.

ATTEMPTS to clarify the boundaries of physician liability for participating in acts that lead to the death of a patient have focused attention on four types of recurring cases, introduced in last month's column.[1] These cases raise issues such as the application of the traditional principle of "double effect"; the provision of drugs having both therapeutic and nontherapeutic uses; the injection of substances designed to end the life of a patient; and direct involvement by physicians in a patient's attempt to commit suicide.

The first type of case involves the application of the principle of double effect. This principle recognizes that an action having both a good and a bad effect is justified only when four conditions have been satisfied: (1) the action itself must not be intrinsically wrong; (2) the actor must intend the good effect alone (though the bad effect may be tolerated); (3) the good effect must be achieved by the action itself, not by the bad effect; and (4) the good effect must outweigh the bad effect risked or permitted.[2] The justification for allowing such actions in a medical context was stated by the President's Commission in its report, *Deciding to Forego Life-Sustaining Treatment:*

> The search for medical treatments that will benefit a patient often involves risk, sometimes great risk, for the patient: for example, some surgery still carries a sizeable risk of mortality, as does much of cancer therapy. Furthermore, seeking to cure disease and to prolong life is only a part of the physician's traditional role in caring for patients; another important part is to comfort patients and relieve their suffering. Sometimes these goals conflict, and a physician and patient (or patient's surrogate) have the authority to decide which goal has priority. Medicine's role in relieving suffering is especially important when a patient is going to die soon, since the suffering of such a patient is not an unavoidable aspect of treatment that might restore health, as it might be for a patient with a curable condition.[3](p79)

The difference between cases justified by the principle of "double effect" and cases involving the injection of substances designed to end the life of the patient was also addressed by the Commission:

> ...the use of pain-relieving medications is distinguished from the use of poisons, though both may result in death, and society places the former into the category of acceptable treatment while continuing the traditional prohibition against the latter. Indeed, in the Commission's view it is not only possible but desirable to draw this distinction. If physicians (and other health professionals) became the dispensers of "treatments" that could only be understood as deliberate killing of patients, patients' trust in them might be seriously undermined. And irreparable damage could be done to health care professionals' self-image and to their ability to devote themselves wholeheartedly to the often arduous task of treating gravely ill patients. Moreover, whether or not one believes there are some instances in which giving a poison might be morally permissible, the Commission considers that the obvious potential for abuse of a public, legal policy condoning such action argues strongly against it.[3] (pp79-80)

Far more challenging than either of the first two types of cases have been the third and fourth types, represented by reports of the acts of Dr. Quill[4] and Dr. Kevorkian.[5] In the case of Dr. Quill, should a doctor be held legally liable if drugs prescribed for a legitimate purpose (to facilitate sleeping) are used by the patient to commit suicide? In the case of Dr. Kevorkian, should a physician who provides the means for a patient to commit suicide be held legally liable if the patient uses those means for that purpose? Discussion of these topics will conclude in next month's column.

REFERENCES

1. Healey J: The emerging legal consensus about end of life decisions. *Conn Med* 1993; 57:345.
2. Beauchamp TL, Childress JF: *Principles of Biomedical Ethics.* New York, Oxford University Press, 1989, p.128.
3. President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research: *Deciding to Forego Life-Sustaining Treatment: Ethical, Medical, and Legal Issues in Treatment Decisions.* Washington DC, U.S. Government Printing Office, 1983.
4. Quill T: Death and dignity: A case of individualized decision making. *N Engl J Med* 1991; 324:691-4.
5. Annas G: Killing machines. *Hastings Cent Rep* 1991; 21:33-5.

JOSEPH M. HEALEY, J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

Correspondents: Joseph M. Healey, J.D., Professor, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington, CT 06030-1910.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

I Swear by Apollo Physician

ROBERT U. MASSEY, M.D.

AS commencement ceremonies at the University of Connecticut School of Medicine drew to a close last month, the new graduates and their older physician colleagues rose and recited together a modification of a World Health Organization's modification of the Hippocratic Oath. We had used it for years, and I thought little about it; it was bland, dull, and politically correct; the words had all but lost their power to move or even to sound serious. A faculty member who had rarely before attended these ceremonies asked afterward, "What happened to the real Oath?" I said that we had never used it, nobody has taken an oath on Apollo for years, and anyway there was that troublesome thing about not giving a woman an abortive remedy. My own attention this year had been drawn rather to the omission of an invocatory prayer, and I reflected on how for so long we had been, in our ignorance and insensitivity, violating the First Amendment; I wondered at the discomfort we had unwittingly inflicted over the years on graduates, their families, and friends by this sneaky effort to establish religion—even though the nondescript prayers had seemed to me inoffensive enough for Deist, Dravidian, or Druid.

On occasions like this we must pause to be grateful for the progress that has characterized these enlightened latter days of the second millennium (Common Era). The Higher Morality, revealed mostly since the 1960s, has convicted us, reminding us in new and unsuspected ways that "we are sinners all."

Some cynics will claim that history has been mostly downhill since the French Revolution, and I confess that when I contemplate the current rage for rights without responsibility, multiculturalism, and all values equal, I am tempted to join this ill-humored crowd. Invocatory prayers are, of course, so much official ritual and for that reason religiously meaningless, but each little thread pulled from our protecting cloak of civility may have far-reaching and quite unpredictable consequences. The fabric of any culture is woven together from thousands of tiny threads of rituals, courtesies, traditions, and rules. In truth, a civilization may be little more than the cloth made from these fibers expressly to cover our shame and restrain our mean, aggressive natures, our only hope of freedom from irrational and arbitrary will, our protection from the abyss.

In the early days of molecular genetics I recall being amazed to learn that the difference between normal and sickle-cell hemoglobin lay in a substitution of one amino acid, the sixth in the beta chain: the replacement of glutamic acid by valine. The clinical, epidemiological, and historical consequences of this seemingly trivial modification of the hemoglobin molecule could never have been predicted. It has long been known by scientists, but usually forgotten by politicians and intellectual social manipulators, that trivial causes may have large, unpredictable, and often malign effects. "For want of a nail the rider was lost." For want of civility, custom, and tradition a culture can be lost, but so gradually as to escape notice.

In the old oath, swearing by Apollo, Aesculapius, Hygieia, and Panaceia made the ceremony, and hence the act, serious by invoking the Guardians, whoever and wherever they might be. To "solemnly pledge," as the greatly paraphrased WHO modification has it, is hardly more than an attempt to seem grave; the idea of a covenant is gone. And by omitting the ritual invocation the transmutation is complete; an ancient oath-taking ceremony is now a mere social event.

This may all be quite unimportant. But if we are concerned, as many seem to be, that the Spirit of the Times, the Zeitgeist, is beginning to look more and more like a mean and shrill destroyer of values and forms that have been around since history was first written, maybe we should hesitate to pick any more threads from the fabric until we have a better sense of the history of this present time. The Zeitgeist may not be as demonic as it seems to me, but only mischievous, noisy, and silly, perhaps a Poltergeist.

ROBERT U. MASSEY, M.D., Professor Emeritus, Division of Humanistic Studies, Department of Community Medicine and Health Care, University of Connecticut School of Medicine, Farmington.

MEDICAL NEWS CAPSULES

This Month's Reading in Review

TIMOTHY B. NORBECK

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

- "If you set up a system...where doctors feel they're imposed upon, particularly because they've not been included in negotiations, you have thousands upon thousands of physicians who have no self-interest in the system working. And that's like having termites in your system."—Former presidential candidate Paul Tsongas urging the Clinton administration to take doctors' concerns into account to make health-care reform succeed.
- The U.S. Public Health Service estimates that lifestyle accounts for 54% of all mortality due to heart disease, 34% of all cancer deaths, 50% of all those due to cerebrovascular disease, and 49% of all deaths caused by arteriosclerosis.
- In a study by Morgan Quitno Corporation of Lawrence (Kansas) based on 22 factors, including percent of population insured, death rates, family income spent on health care, and per capita rates of smoking, drinking, and overeating, Utah (with a 73% Mormon population which eschews alcohol and tobacco) was considered as the healthiest state with its neighbors, Idaho and Wyoming, placing second and third, respectively....Connecticut placed 19th overall while Florida finished dead last.
- A new Louis Harris poll on American health habits finds increasing numbers of people are eating more of the wrong foods, carrying too much weight, speeding when driving, and generally doing a poorer job of taking care of themselves than they were just a few years ago....The president of the polling firm said that "it's quite likely the healthier lifestyles of the 80s were just a passing fad."
- Seven studies done all over the world show that the more education a person has, the less likely that he or she will suffer from memory-robbing disorders such as Alzheimer's disease or other forms of dementia....The most dramatic of the seven studies was one on more than 5,000 Shanghai residents 55 to 75 years old....All other factors being the same except learning, those with no education had twice the risk of being demented as those with elementary or middle-school education.
- Florida is facing a quiet catastrophe as more of its population develops Alzheimer's disease....The number of Alzheimer's cases in Florida is expected to double in the next eight years from its present 290,000 cases to 576,000.
- Ofttimes physician and health care critic, Lee Iacocca, has boasted that Chrysler's new Concorde ushers in "the renaissance of the American car"....He doesn't bother to mention that the cars are assembled in Canada with 28% non-American parts.
- An Employee Benefit Research Institute (EBRI) poll indicated that 83% rated the quality of health care they had received in the past year as excellent or good....But only 27% of those same respondents rated the country's health care system as excellent or good.
- According to the General Accounting Office (GAO), a new report shows that 43% of the 99 million patients seen in emergency rooms in 1990 had "minor ailments that could have been treated elsewhere"....GAO also found that 60% of the non-urgent cases in emergency rooms had primary care physicians but didn't use them.
- Tobacco, alcohol and illegal drug-related illnesses and injuries accounted for 28% of the admissions and 39% of the medical costs to the Johns Hopkins Hospital intensive care unit during a 15-week period in 1989....The study, which appeared in January's issue of the journal *Chest*, cautioned that although Hopkins is an inner-city hospital and serves a broader community, its findings do not necessarily apply to other hospitals, "but it is a place to start examining the hidden costs of substance abuse."
- According to the Census Bureau, $40 billion per year is spent on illegal drugs, only a little less than the federal government's education budget.
- "You and your habits, not the doctors, are the single biggest problem in this country."—Columnist Mike Royko in writing what the Clinton administration "should have told the American public."
- *Only in America:* In June of 1992, the New Jersey Department of Community Affairs ordered all of the state's 567 towns to provide for elevator inspection service. A spokesman said the state cannot make exceptions to rules, otherwise the "Uniform Construction Code would no longer be uniform." Therefore, three New Jersey townships—Upper Pittsgrove, Alloway, and Quinton—have recently contracted to have elevator inspectors. There is not a single elevator in any of those three municipalities.

TIMOTHY B. NORBECK, Executive Director, Connecticut State Medical Society.

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgement), provided that the are submitted in duplicate, signed by all authors, typewritten in double spacing, and do not exceed 1-1/2 pages of text (excluding references). They should not duplicate similar material being submitted or published elsewhere. Letters referring to a recent Journal article should be received within six weeks of the article's publication.

MEDICAL HYDROLOGY

To the Editor: I am writing you regarding the February 1993 article in Connecticut Medicine which I read with great delight. As former director of the American Society of Medical Hydrology which is a special interest group of the American Academy of Physical Medicine and Rehabilitation, and meets annually with the Academy, I believe that with the mineral water resources in the U.S., we are able to upgrade and revive our mineral water baths and treatments. My interest in medical hydrology began while I was a graduate student at Yale University. Since then I have organized educational trips for American physicians and health professionals to medical congresses and European spas to learn first-hand about medical hydrology.

The American Society of Hydrology (ASMH), is a small society of physical medicine physicians which was formed in 1960 by the late Sidney Licht, M.D., who was director of physical medicine and rehabilitation at Yale School of Medicine. He sincerely believed there was a need to incorporate and utilize the mineral waters in the U.S. for physical therapy and thus wrote a medical textbook in 1963 entitled *Medical Hydrology* which has become one of the only sources written in English for the use of water in physical therapy. I have undertaken the revision of this book.

Nevertheless, the English-speaking countries of the world have underestimated the use of hydrology in physical medicine whereas in Europe and much of the rest of the world the use of water in treating arthritis, back injuries, skin diseases, etc. is the cornerstone of physical medicine. However, the use of water is making a comeback in American physical medicine with aqua exercises at YWCA and YMCAs and revival of spa therapies at American spas. But one crucial issue is at hand, the thermal mineral waters along with their steam and vapors are not utilized. The waters are the main therapeutic aid of which the majority in the U.S. are left unused. Saratoga spa enjoyed its heyday in the early 1900s offering superb mineral water for treatment of arthritis and digestive ailments; now the bath houses are in disrepair and the water flows with no one benefiting from its therapeutic properties.

My point is that American spa medicine should be organized and comparable to that found in Europe. There are so many wonderful springs, such as Hot Springs, Arkansas; Warm Springs, Virginia; etc. to be taken advantage of. It would be enlightening to compare some of the French and other European spas to those found today in the U.S.

Margaret L. Pearson, M.P.H.
Ex-Officio Director
American Society of Medical Hydrology
Southbury

IN RESPONSE TO DR. VOLPINTESTA

To the Editor: The April 1993 issue contains a letter from Dr. Volpintesta sharply questioning Dr. Ahamed's recent column about the "continuous communication taking place in his hospital between physicians and administrators aimed at improving hospital-staff relations." Dr. Volpintesta challenges Dr. Ahamed because he believes "such interactions are often unproductive" and demands an explanation.

Besides finding it somewhat hard to believe that one could attack and challenge someone of Dr. Ahamed's stature, I am pleased to answer some of the writer's questions.

On our governing board, four of the 18 members are practicing physicians with full voting privileges. Their contributions count for a good deal. The Vice President for Medical Affairs also attends these meetings. The Staff Relations Committee, a committee of the hospital, is chaired by a practicing physician and its membership includes three others. Every committee of the hospital contains at least one member of the medical staff and in the various corporate structures there are physicians in every branch and, in at least one instance, chaired by a physician.

The President/CEO attends Medical Executive Committee and Medical Staff meetings and reports at each one. He also meets at regular intervals with the Vice President for Medical Affairs, the President of the Medical Staff, and the Chairman of the Medical Executive Committee. In addition, there is constant traffic by members of the Medical Staff through the Medical Staff Office and the office of the President/CEO.

While interests of medical staff, hospital administration, and governing body inevitably diverge at times, I think most hospitals are now following the patterns described above with constant interchanges of information, ideas, and suggestions.

Surely Dr. Ahamed would never have offered such information unless it were true and carefully thought out.

Harvey N. Mandell, M.D.
Vice President for Medical Affairs
William W. Backus Hospital
Norwich

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

Fairfield County Medical Association
Hartford County Medical Association
New Haven County Medical Association
New London County Medical Association
CNA Insurance Companies

We're proud of the company we keep

© 1993 Poe & Associates, Inc.

Since 1981, each of these important associations has endorsed and supported the Physicians Protector Plan®, the finest professional liability insurance program available in Connecticut today.

This 12-year working relationship among the medical community, the program administrator, and CNA Insurance Companies has provided physicians in Connecticut with unsurpassed commitment, expertise, financial stability, and a stable professional liability environment.

We at PHYSICIANplans® are proud to be associated with Connecticut's medical community and with CNA. If you're not currently participating in this fine program this is an excellent opportunity to discuss how it can benefit you and your practice. Give us a call.

1-800-533-4539

PHYSICIANplans®

CNA
For All the Commitments You Make®

The Physicians Protector Plan® is a trademark of Poe & Associates, Inc., Tampa, Florida, and is underwritten by the Continental Casualty Company and National Fire Insurance Company of Hartford, two of the CNA Insurance Companies.

From the Executive Director's Office

COUNCIL MINUTES

Wednesday, 7 April 1993

Attendance

Present in addition to the chairman, Dr. Joseph Czarsty were, Drs. Ahamed, Beck, Blum, Bobruff, Brooks, Deren, Eslami, Fischbein, Freedman, Kamens, Keating, McDonnell, Orphanos, Parke, Petro, Redmond, Sadowski, Schwartz, Tesoro, Timmerman, and Zanker.

Also present were: Mr. Norbeck, Mrs. Lindquist, Mr. Brunell, Mrs. Connelly, Mrs. Sullivan, Mr. Garofalo, Mr. Thompson (FCMA), Mr. Freberg (NHCMA, Mr. Coffey (HCMA), and Mrs. Czarsty.

Absent were: Drs. Cottle, B. Fox, Franklin, Hollister, Hoskings, Leach, Lesnik, Meridy, Sosa, Van Nostrand, Wetstone, Wolfson, and Zeppieri.

Reports of Related Organizations

CPRO: Dr. Kamens reported that CPRO has been going to hospitals giving data as a result of the studies made on mortality and stated that the reception has been enthusiastic.

Dr. Kamens further reported that he represented CSMS at the Uniform Clinical Data Set (UCDS) meeting sponsored by the AMA, and stated that the physicians attending were very much in tune in being able to write their own criteria and parameters. The meeting was well received.

Report of the President

(a) *Building Renovation:* Dr. Ahamed outlined the background leading to the selection of ACMAT Corporation to make a feasibility study of the renovation of the building following which representatives of the ACMAT Corporation presented sketches of the proposed renovations and proposed costs. After full discussion, including parking, additional space, etc. It was VOTED to approve the project.

(b) *Liaison Committee with Hospital Medical Staffs:* Dr. Ahamed reported that he felt that it was important for CSMS to develop a liaison with hospital medical staffs of the Connecticut general hospitals. CSMS should have a physician from each hospital representing CSMS on the medical staffs and that person should request the president of the medical staff to provide an opportunity on the agenda for the representative to give a report. This would be an expeditious way to transmit information on CSMS activities to hospital medical staffs. It was his suggestion that the representatives should be members of the Council or House of Delegates.

Representatives who are not on the Council could request information prior to each staff meeting. It was VOTED to create a Liaison Committee to interact with the hospital medical staffs of the Connecticut General hospitals. A list of Connecticut Hospitals was circulated with the request that volunteers sign up to serve as liaison with the various hospitals.

(c) *Connecticut Hospital Association Meeting:* Dr. Ahamed reported on a meeting which he attended, along with Drs. Meridy, Betton, Kamens and Mr. Brunell of the CSMS staff, on the subject of publishing data on hospitals and individual physicians. This data would involve procedures performed, mortality statistics, length of stay in the hospital, etc. Dr. Kamens expressed concern about confidentiality and accuracy of data. Mr. Brunell reported that CHA would be inviting all the councilors to a meeting at which this subject will be discussed. It was VOTED that Drs. Betton, Kamens, Meridy and two other appointees act as nominees to the CHA Committee on Quality Assessment

(d) *Meeting with Public Health Committee:* He reported that he participated in a round table discussion with the Public Health Committee, which included representatives from the insurance industry, HMOs, labor, business, CCAG, and AARP concerning health care reform.

(e) *Unified Membership:* Dr. Ahamed reported that since the last meeting he attended the Fairfield County Medical Association Board meeting and was pleased to say that they voted in favor of unified membership. The three large counties have now voted in favor of unified membership and he stated that hopefully 1993 will be the year that unified membership will be accomplished. It is his intent to attend as many of the county meetings as possible to discuss the issue.

(f) *CMIC:* He reported that at the last CMIC board meeting they voted to reduce the payment to the county associations for their efforts on behalf of CMIC, and that he has received a great many calls from the counties protesting this action. At the present time CMIC is paying $125 per insured to the counties. He stated that he is going back to the board of CMIC to ask them to reconsider their action.

(g) *Governor's Task Force on Sexual Violence:* A letter was received by Dr. Ahamed from the Chairman of the CSMS Committee on Physician Health urging the Council to seek significant representation on any Task Force formed to examine the report "Sexual Violence in Connecticut" which has been published by the Governor's Task Force. It was VOTED that the Chairman of the Council would pursue this matter. The names of Drs. Hollister and Redmond were recommended.

Dr. Ahamed reported that he has not been able to implement all of the ideas he had when he took over the presidency of CSMS and plans to pass those he could not implement to the incoming president. He stated that CSMS has to make every effort to involve young physicians in organized medicine at the local, state and national level to insure the future stability and growth of the organizations, and that CSMS should groom some young physicians as AMA delegates and promote their growth at national level. He further suggested that we should trust and be supportive of the leadership of the various organizations that CSMS has created. Since this would be his last Council meeting as president, he expressed his thanks and appreciation to members of the Council for the encouragement and cooperation that they have given him. Dr. Ahamed was given a standing ovation.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Report of Executive Director

Mr. Norbeck reported that he participated in a two and a half hours panel discussion on health care reform with representatives of Cigna and CCAG at the Hartford Diocese meeting of the Board of Trustees of the Connecticut Interfaith Housing and Human Services Corporation. Cynthia Mathews, Connecticut Commissioner on Aging moderated.

The issue of mandatory assignment resurfaced and Mr. Norbeck informed the Council that he spoke against the issue at a public hearing held by the Human Services Committee on March 16th. It is his feeling that mandatory assignment will be defeated.

On March 29, Mr. Norbeck participated in another panel discussion on health care reform at the Hartford Medical Society. Other panelist were IAC President Bob Kehmna, CHHC Commissioner Don Pogue, and Dr. Paul Dolinsky. Dr. Robert Massey was the moderator. Mr. Norbeck informed the Council that we should participate in as many of these panel discussions, etc. as possible so that we get the message out to the people.

Mr. Norbeck reported on the AMA Washington meeting, held on March 24th and 25th, to meet with the legislators from the various states. Drs. Bobruff, Hollister, Wolfson, Mr. Freberg (NHCMA), Mr. Thompson (FCMA) attended from Connecticut. It was a very worthwhile session and individual meetings had been set up with the Connecticut legislators and the CSMS representatives. A summary of the meeting, prepared by Mr. Norbeck, will appear in the CSMS newsletter.

Report of the Auxiliary

Mrs. Joseph Czarsty reported that the Auxiliary had its last meeting at which time they closed their books. She reported that representatives from all of the counties attended the meeting. They voted to disburse their funds as follows: $5,000 to Middlesex County Auxiliary as they are going to continue their county association and will be the conduit into Connecticut from the national office; $1,000 each to Windham County and New Haven County for scholarships and $10,000 to CSMS to be used for the pre-immunization program. She further stated that she hoped that CSMS would continue to give to the AMA-ERF program by raising funds through its billing process.

Legislative Update

Councilors were informed that CSMS was sponsoring a "Healthy Breakfast on the Hill" which was to be held on April 14 at the Capitol. They were urged to take advantage of this opportunity to meet their legislators and discuss issues involving health care.

An update on pertinent state legislation was distributed to members of the Council which discussed health care reform bills, Workers' Compensation reform, licensing of x-ray technicians, and Medicaid physician reimbursement. Testimony was presented on these bills at the various hearings held. Lengthy discussion pursued concerning the x-ray licensing bill and it was VOTED that the CSMS position should be that the physician or his/her designate under their supervision should be allowed to take x-rays in their office.

ACCUMED

Jeannette Schreiber, Esq., who has been working on the Workers' Compensation issue, joined the Council to give a progress report. She stated that it is expected that a final report for submission to Commissioner Frankl would be accomplished within a few more weeks. The report would include the work that the advisory group has done, the proposed regulations that outline the new mechanism for establishing fees, guidelines as to how some prior outstanding claims are to be resolved, and some proposed detail billing guidelines. It will then be up to Commissioner Frankl to adopt these proposals, which have been approved by CBIA, IAC, CSMS and other members of the task force that have been working on this issue. The Commissioner will have to resolve a few open issues including establishing what the uniform percentile per payment will be based on the ranges that have been proposed by the various participants in this study. Also a proposal is to have a required annual update, subject to an inflationary cap. She reported that the advisory committee has essentially accomplished what they set out to do, which was to establish a system that has uniformity, predictability and a fair fee. It is hopeful that the Commissioner will accept the proposals and proceed to put them into regulations. She further reported that the legislature will be mandating a physician fee schedule, along with an attorney fee schedule, for Workers' Compensation. The CSMS lobbyist has been working on the language of the bill and to assure that the fee schedule is consistent with the one proposed by the advisory committee.

Report on CMMI and CMIC Activities

In accordance with Council action, prior to each House of Delegates meeting, there should be a discussion and report on CMMI and CMIC. Dr. Eslami, Chairman, submitted a written report which reviewed a report on CMIC Board actions and company operations for 1992. It was VOTED to receive the report as information.

Report of Pension Administrative Agency

In accordance with Council action, prior to each annual meeting of the House of Delegates, the Pension Administrative Agency, there should be a report to the Council. Dr. McDonnell reported that all directives of the Pension Administrative Agency have been followed and all necessary reports have been filed and that the pension obligations are fully funded. It was VOTED to receive the report as information.

Workers' Compensation Commission

Jesse H. Frankl, Chairman of the Workers' Compensation Commission, joined the Council to discuss the subject of "Physicians' Education under the Workers' Compensation Commission." Commissioner Frankl reported that he had made several changes in the Workers' Compensation administration pursuant to a Public Act that was passed in 1991. He stated that he believed that physicians were the most vital individuals in the system and nothing has been done with regard to the handling of Workers' Compensation cases. He reported that he had developed a video covering this subject and provided each councilor with a copy of the video to take back to their office for their review (which should include their office personnel).

Commissioner Frankl became chairman on June 1, 1992 and one of his duties was to implement the Public Act passed in 1991. One of first things he did was put together a Medical Advisory Panel as required by the statutes. He reported that as a result of meeting with the Medical Advisory Panel, which is a nine member panel, composed of physicians from all over the state and also includes a hospital administrator, dentist, president of the Chiropractic Association, a neurosurgeon, orthopedist and pyschiatrist, they have perfected regulations dealing with preferred provider plans and the regulations have been approved by the Connecticut Legislature and submitted to the Secretary of State and he is now accepting plans for review. Physicians who see Workers' Compensation patients may be approached by various employer groups regarding these preferred provider plans. He stated that it is his intention and number one priority to arrange meetings with physicians in the various county medical associations to go over procedures done in Workers' Compensation cases. The presentation was followed by a discussion session.

Life Members

It was VOTED to receive as information the name of the following physician who was granted Life Membership:

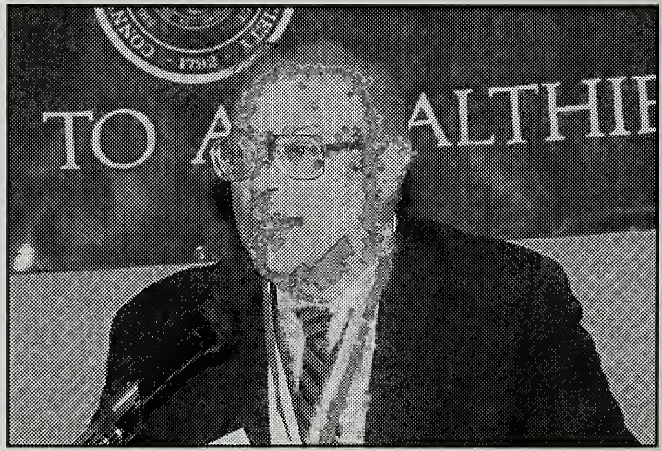
Edwin D. Niesyn, M.D., George's Mills, New Hampshire (NH).

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

R. William Alexander, M.D., Member AMPAC Board of Trustees, presented AMPAC Achievement Award to Stewart J. Petrie, M.D., COMPAC Chairman.

Joseph C. Czarsty, M.D., Chairman of CSMS Council, presented Past President's pin to Sultan Ahamed, M.D.

Sultan Ahamed, M.D., giving Oath of Office to new president, Howard J. Wetstone, M.D.



Howard J. Wetstone, M.D. giving his inaugural address.

Sultan Ahamed, M.D. presented award to James P. Orphanos, M.D., who retired as Alternate Delegate to AMA.

John P. Bigos, M.D., Councilor from New London County; Palma E. Formica, M.D., Member AMA Board of Trustees; and Anthony P. Redmond, M.D., Alternate Delegate to AMA.

SUMMARY OF PROCEEDINGS
CSMS HOUSE OF DELEGATES — ANNUAL MEETING
RAMADA INN, MERIDEN — MAY 12, 1993

Reports and Addresses

The House received reports, addresses and/or remarks from the President, Secretary, Treasurer, the Chairman of the Council, Delegates to the AMA, Chairman of the Board of Directors of COMPAC, the CSMS Executive Director, M.D. Health Plan, Connecticut Medical Management, Inc. (CMMI), Standing Committees, Representatives and Advisors, Auxiliary to CSMS and Delegates from the Medical Society of New Jersey, Massachusetts Medical Society and Medical Society of the State of New York. All of the reports were published in the Handbook that was distributed to the delegates, with a few exceptions. The address of the retiring President, Sultan Ahamed, M.D. and the Executive Director, Tim Norbeck, appear at the conclusion of this summmary. The inaugural address of the new President, Howard Wetstone, M.D., appears as the President's Page in this issue.

PRINCIPAL ACTIONS TAKEN

Election of Officers and Others

The House voted to approve the published report of the Nominating Committee (the Council). Officers elected were:

President: Howard J. Wetstone, M.D. Hartford President-Elect: Theodore Zanker, M.D., New Haven Vice-President: Dickerman Hollister, Jr., M.D., Greenwich Secretary: Marjorie G. Petro, M.D., Willimantic Treasurer: Robert R. McDonnell, M.D., New Haven Speaker of the House of Delegates: Michael M.Deren, M.D., New London Vice-Speaker of the House of Delegates: Neil H. Brooks, M.D., Rockville Councilor-at-Large: Sultan Ahamed, M.D., Norwich Delegates and Alternates to the American Medical Association:

Edward A. Kamens, M.D. Fairfield (D)
  1/1/94-12/31/95
    Joseph C. Czarsty, M.D., Waterbury (A)
      1/1/94-12/31/95
Jerome Bobruff, M.D., New London (D)
  1/1/94-12/31/95
    Roger S. Beck, M.D. Wethersfield (A)
      1/1/94-12/31/95
Jerome K. Freedman, M.D., Branford (D)
  1/1/93-12/31/94
    Neil H. Brooks, M.D., Rockville (A)
      1/1/93-12/31/94
Joseph S. Sadowski, M.D., Hartford (D)
  1/1/93-1/1/94
    Anthony P. Redmond, M.D., Greenwich (A)
      1/1/93-12/31/94
Mehdi S. Eslami, M.D., Waterbury (D)
  1/1/93-12/31/94
    John B. Franklin, M.D., Hartford (A)
      1/1/93-12/31/94

In approving the report of the Nominating Committee, it was also voted to combine the Committee on Third Party Payments with the Liaison Committee with the Department of Income Maintenance; disband the Committee on Cooperation with the Medical Schools of Connecticut; discontinue Advisors to the Auxiliary to CSMS; and change delegates to Massachusetts to President (D) and President-Elect (A).

Resolution on Reduction in Reimbursement from CMIC

It was voted to table the subject resolution submitted by the Fairfield County Medical Association.

Resolution on Mandatory use of Helmets by Motorcyclists

It was voted to approve the following resolution submitted by the Fairfield County Medical Association:

RESOLVED, that the Connecticut State Medical Society shall continue to strongly advocate for the passage of a law which would require the wearing of a safety helmet by motorcyclists of all ages.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Resolution on the Release Physician Specific Data

It was voted to approve a resolution submitted by Jerome K. Freedman, M.D., New Haven, with amendments and which reads as follows:

RESOLVED, that CSMS believes that any hospital specific and/or physician specific data that may be released by the Connecticut Hospital Association to the public must be not only valid data, but also adjusted for severity of illness, and be it further

RESOLVED, that CSMS believes that the data should be collected prospectively, compared to a national data base, and independently audited before release.

AWARDS

American Medical Education and Research Foundation (AMEF)

Dr. Ahamed presented the following awards:

University of Connecticut School of Medicine... $5,416.25—Peter Deckers, M.D., Interim Dean, accepted the check.

Yale University School of Medicine also was granted $5,453.25—and the check will be presented at a future date, since the Dean was unable to be present.

Connecticut Science Fair Awards

Dr. Ahamed announced the following awards:

First Place—Senior Division — Mr. Carlos M. Toro, Hartford, Hartford Public High School

Project: "The Metallic Epidemic"

Second Place—Senior Division — Miss Jennifer A. Palacio, Hamden, Hamden High School

Project: "Yuck! What Is That Smell?"

First Place—Junior Division — Miss Melissa M. Levack, Portland, Independent Day School, Middlefield

Project: "Antibiotic Resistance and Responce to Varied Therapy"

Second Place—Junior Division — Miss Tanya M. Markiewicz and Miss Meredith M. Renda, both of Woodbury, Woodbury Middle School

Project: "What are the Effects of Measured Doses of Sodium Fluoride on D. Melanogas?"

The first place winners received a plaque and a check. The second place winners received a copy of the AMA's Family Medical Guide.

Award to Mrs. Roberta Czarsty

Dr. Ahamed awarded a certificate to Mrs. Czarsty as President of the Auxiliary, in recognition of her dedicated leadership in the work of the Auxiliary and also commended the Auxiliary on its completion of fifty years of service and charity.

It was announced that after 50 years the Auxiliary will be disbanding. Everything possible was tried to keep the Auxiliary going, without success. The funds were distributed as follows: $5,000 to Middlesex County, with 71 members, as they are going to continue their county association and will be the conduit from Connecticut to the national office; $1,000 each to Windham and New Haven Counties for scholarships; and $10,000 to CSMS to be used for the pre-immunization program.

COMPAC AWARD

Dr. R. William Alexander, member of the AMPAC Board of Director, addressed the House and stated never was membership in the local PACS and AMPAC more crucial. He urged past members to renew their membership and urged the promotion of new members. He stated that PAC membership will reap benefits for the physician and the patients for years to come. He presented Stewart J. Petrie, M.D., Chairman of COMPAC, with the AMPAC Achievement Membership Award, which was granted for increasing membership in COMPAC and AMPAC.

Award to Retiring Delegates to the AMA

Dr. Ahamed paid tribute to Stewart J. Petrie, M.D. and James P. Orphanos, M.D. for their service as delegates to the AMA. Dr. Petrie has been a member of the delegation from 1981-1992 and served as chairman. Dr. Orphanos has been a member of the AMA delegation from 1987-1992. He stated that both of these men have distinguished themselves in their profession and have given unselfishly of themselves to organized medicine. Both were presented with an inscribed Paul Revere silver bowl.

Award to Retiring CSMS Employee

Mr. Tim Norbeck, Executive Director, presented an appropriately inscribed certificate and silver bowl to Dorothy Stratton, who retired on April 30, after 38 years of service with CSMS. Miss Stratton was responsible for all of the membership records. Mr Norbeck stated that Miss Stratton epitomized loyalty and dedication to the staff with whom she worked and the doctors for whom she worked.

Award to Retiring President

Dr. Joseph Czarsty, Chairman of the Council, presented a Paul Revere silver bowl and pin to Sultan Ahamed, M.D.

Inauguration of New President

Dr. Ahamed gave the Oath to the new president, Dr. Howard J. Wetstone of Hartford and presented him with a presidential medallion, with his name and date inscribed on it and a gavel.

Keynote Address

Dr. Palma E. Formica of Old Bridge, New Jersey, a member of the AMA Board of Trustees, addressed the House. She spoke about the AMA's involvement in the Health Reform Bill and informed them that the AMA was keeping a constant vigilance on activities taking place in Washington.

The meeting concluded with a reception for the new President.

Collegial Greetings from Massachusetts

LEONARD J. MORSE, M.D.

Dr. Ahamed, Dr. Wetstone, Dr. Deren, Officers and Members of the Connecticut State Medical Society:

I am very pleased to convey best wishes to you from Dr. William Callahan, President, and the other officers of the Massachusetts Medical Society on the occasion of the Connecticut State Medical Society's 201st annual meeting of the House of Delegates. Significant happenings in Massachusetts relating to the noble profession of medicine are similar to those happening in Connecticut and elsewhere in these United States.

We have the privilege of living in the epicenter of the world's best medicine. We have a duty to maintain and preserve that which must be preserved. I believe freedom of choice—freedom for patients to seek the services of a doctor of their choice and freedom to select a health plan of their choice must be preserved. Representative Peter Stark (D-CA), perhaps medicine's most vociferous critic, addressed those 1,000 physicians gathered in Washington, D.C., last month at the A.M.A. meeting entitled "A Time for a New Partnership." He said, "My mother will not be happy if she cannot choose her own doctor"!

Managed competition, in my opinion, is an oxymoron; what we need is managed cooperation. Exclusive contracting by physician hospital organizations and health insurance companies is undermining the significance of a medical license and inhibiting physicians having laboriously earned credentials from serving those in need, and from earning a living.

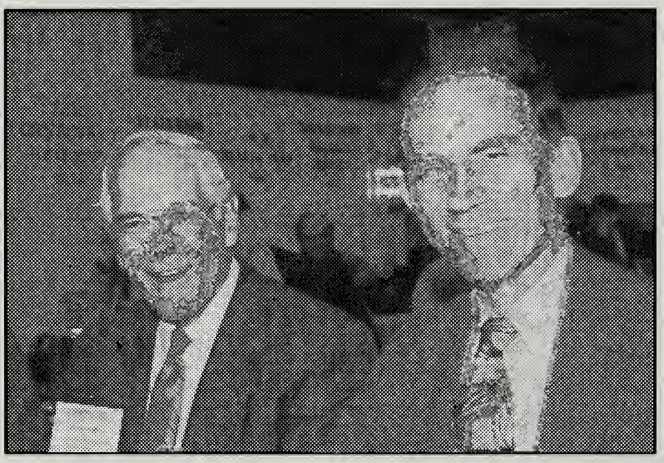
We look forward to change—health care reform—with anticipation that things will improve, not worsen. Vice President Albert Gore told us that if the doctors of America cooperate in attempting to lower the cost of health care, we can anticipate medical liability reform and the de-obfuscation of our office management. It is a credible exchange. In conjunction with the A.M.A. leadership and Health Access America, we all should support the sculpturing of health care reform. Thank you.

Dr. Morse is President-Elect of the Massachusetts Medical Society, and practices internal medicine/infectious disease in Worcester.

Tim Norbeck, Executive Director, Dorothy Stratton, CSMS Staff, and Josephine Lindquist, Associate Executive Director. Ms. Stratton was presented an award on her retirement after 38 years of service in the membership department.

Howard W. Meridy, M.D., Councilor and Leslie P. Goldman, M.D., Delegate, of Litchfield County Medical Association.

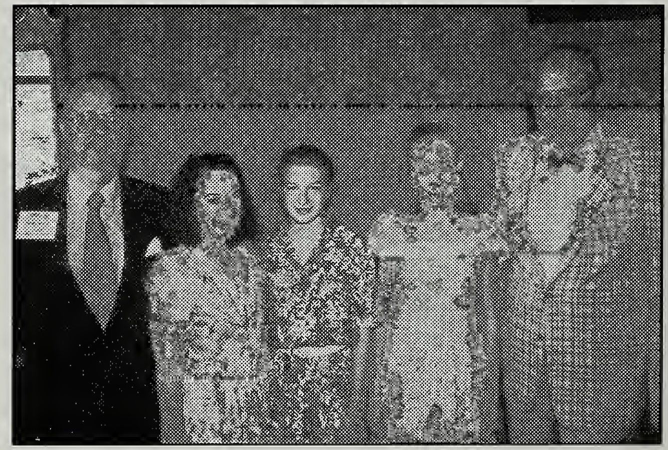
HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE



Roger S. Beck, M.D., Alternate Delegate to AMA, and Alfred Herzog, M.D., Immediate Past-president, of Hartford County Medical Association.

Vincent J. Catrini, M.D., Chairman of Board, and Mahmoud Okasha, M.D., Member, Board of Directors, both of M.D. Health Plan.

Mr. Gary Fredberg, Executive Director, of New Haven County Medical Association, and Theodor Zanker, M.D., President Elect, CSMS.



Meredith M. Renda, Tanya M. Markiwicz, both of Middlebury Middle School, Melissa M. Levack of Independent Day School, Middlefield, Science Fair Winners pictured with judges Michael M. Deren, M.D. and W. Raymond James, M.D.

Address of the Retiring President

CSMS Annual Meeting—House of Delegates
12 May 1993

SULTAN AHAMED, M.D.

IT has been a very exciting year. When I assumed the office of the president of CSMS, I was apprehensive and uncertain. But with the help of the staff and so many of you, we have accomplished a lot in the past year. A lot more needs to be done in the future under the able leadership of of your successive presidents.

Albert Camus said " Politics and the fate of mankind are shaped by men without ideals and men without greatness." I am afraid that the new health care reform that is to be proposed will fit that statement.It will be all encompassing and will affect us all.That proposal will be debated at the congressional level when the input from the various groups will be taken in to account. What ever happens, I am sure the house of medicine will have a say in that outcome. To facilitate the ongoing discussion about the health care, it is very important for the doctors of the state and the country to do certain things so that nothing is railroaded through.

1. Let us inform the public, our patients, the true reason for the raising health care cost.

2. Let us make everyone aware what it takes to become a physician- thirteen to fifteen years of education after high school and 70 to 80 hour work weeks and more than $100,000 in debt before we start our practice.

3. Let us inform the public about the number of hours we work, the number of patients we see and the amount of money we make when compared to other professions. Let us tell them that we are compensated well but we should be. Minority of doctors who make a lot of money does not constitute the average income. If we are given authority and given relief in the antitrust laws the organized medical societies will be able to handle the price gougers.

4. We have a blueprint for health care reform. The AMAs Health Access America is a model proposal. Let us make that and our own reform proposals available to the public and the politicians.

5. Let us make sure that the public understands the evils of socialized medicine. When the whole world is turning away from socialism we should not be replacing a fine medical delivery system with a socialized medical system.

6. Let us proclaim that we will not tolerate slippage in the quality of care by the buzz words of managed competition, expense target, global budget etc.

7. Let us stand on the side of our patients and fight for them against the insurance industry and government bureaucracy.

8. Good and compassionate health care needs choice for the patients. Discrimination by HMOs and insurance companies against certain physicians or the hospitals impedes that choice. we must try to achieve an anti-discrimination law at the state level.

Now let me look back at this past year and compliment the Council and this House and the rank and file members for the accomplishments of our Society. We created a position paper on health care reform. We have had enormous success with the workers compensation commission and the insurance companies. We have ongoing dialogue with the Department of Income Maintanance and the Medicare intermediary. We have helped the worthy causes through our charitable trust. We have had a good year in the legislative arena. We have started a new renovations project of our CSMS headquarters to make it functional into the next century. We are actively involved with CHA and working as a watchdog in their release of doctor specific morbidity and mortality data to the public.

Let us not rest on our past achievements. As Thomas Fuller, the English physician and writer said n If you count every step, you will make long journey of it." So let us hurry along with the purpose in our mind and the eyes on the prize. I request all of you to work vigorousely for achieving unity among the doctors. Unity between the generalist and the specialist, unity between the employed and the fee for service doctors and unity between the university and the community physicians is an absolute must to advance the cause of medicine. Let us put our differences down and unite to achieve what we want and need. The only umbrella organization to carry the torch and march forward is the State Medical Society. If we are disorganized it is easy for the external forces to pick us individually apart. I request all of you to work toward an unified membership of the state and county organizations.

Finally I want to thank the staff for the wonderful support I received during my presidency. Tim Norbeck and his staff are super people who work tirelessly for the doctors of this state. I want to thank the Council members for their cooperation and advice and my county medical association director Art Schuman and my county board for their unfailing support. Last but not least I want to thank my lovely wife Ann and my daughter Chandra for putting up with many absent evenings and days from home and for their patience and understanding.

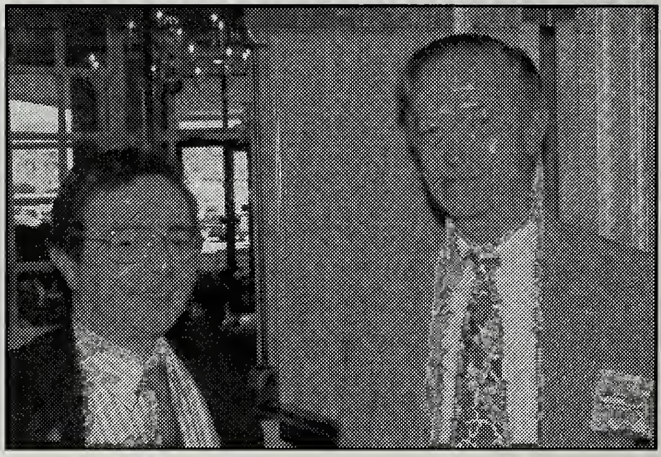
As I leave this position I would like to leave you with the verses from Tennyson's poem about Ulysses, the Greek hero :

How dull it is to pause, to make an end
To rest unburnished, not shine in use!
To follow knowledge like a sinking star
Beyond the utmost bound of human thought
Come, my friends
'T is not too late to seek a newer world.
That which we are, we are
One equal temper of heroic hearts,
Made weak by time and fate, but strong in will
To strive, to seek, to find and not to yield.

Thank you very much.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Edward Kamens, M.D., Delegate to AMA and Neil H. Brooks, M.D., Alternate Delegate to AMA.



Stephen R. Katz, M.D., Delegate, and Robert D. Cottle, M.D., Councilor, of Fairfield County Medical Association.

Mag Connelly, CSMS Staff, Dickerman Hollister, Jr., M.D., Vice-President, CSMS.

Joseph M. Misiak, M.D., Delegate, of Hartford County Medical Association, and Andrew C. Wormser, M.D., Delegate, of New Haven County Medical Association.

Address of the Executive Director

CSMS Annual Meeting—House of Delegates
12 May 1993

TIMOTHY B. NORBECK

IT was a muggy July 2, 1881 when U.S. President James Garfield was about to step onto a Washington, D.C. train in order to attend the commencement exercises at his alma mater, Williams College. A short, thin man pressed forward and fired two shots point-blank at the president, the first striking Garfield in the back while the second barely missed his arm. An operation to remove the bullet was deemed impossible and President Garfield survived for 11 weeks with the bullet lodged behind his pancreas before dying on September 19.

The assassin, Charles Julius Guiteau, was convicted of the crime and hanged the next year. But right up to the gallows he disavowed any responsibility for having killed the president. "I didn't kill him," he said, "the doctors did that. I merely shot him".

This is a rather tame response, of course, compared to what some members of Congress and the public are saying today. As you know, there has been a concerted effort in the past two months to discredit physicians for making too much money. It began with the charge by Families USAs Ron Pollack and then has spread throughout the country with the help of the media. There is a growing number of people who feel that the U.S. Government should limit how much doctors can charge for medical care.

Forget that there are already many inherent costs limiting mechanisms in the system now; 67% of the public favor such limits on all your charges in the latest poll. Others range up to 84% approval for these limits. No doubt they would include your costs plus a tiny stipend. No one seems to understand that physician fees are not physician incomes.

They don't recognize that after your practice expenses and overhead, physicians only receive about 9% of the health dollar before taxes. A Families USA survey indicated that the public thinks that physicians should be limited to $80,000 annual incomes. The telephone pollster asked a thousand people what they thought anesthesiologists and radiologists earned. Of course, the simplistic language of the survey—"Anesthesiologists are doctors who put patients to sleep and numb areas of the body before surgery"—practically invited respondents to lowball the value of what these physicians do.

Families USA didn't think it was worth mentioning that behind that one sentence job description were four years of college, four years of medical school, another four years of internship and residency training (five for radiologists) and fellowships for some—or that physicians, on average, have a 50% longer work week than the average American.

The public, of course, has not been asked by the media what an acceptable income for journalists might be or to agree with the assertion that any reporter, anchor, announcer or editor who makes more is a charlatan or a crook. Should a physician be limited to costs plus a tiny profit for excising the ruptured appendix of a TV anchor who earns $400,000 a year in salary and gets $15,000 to $20,000 for a 30 minute speech?

As columnist Carl Rowan has said: "We will diminish medical care in America if health reform becomes a "level-everybody-to-one-class" assault upon the truly honest, dedicated medical people who earn and deserve compensation far beyond what an uninformed public says is a "reasonable average".

Furthermore, physicians cannot and should not have to function in an environment where they are completely denied the most basic right of the American economic system—to participate in the process through which service price is established.

Perhaps some of those doctor bashers in Congress should look in the mirror and see themselves for what they are: "They came to Washington to do good.... and stayed for years to do well."

Many physicians are concerned about the health reform package, partly because of the 133 full-time government employees who work for members of Congress or for congressional committees and who are members of the 511 member health panel planning the president's reform measure. Historically, such staff have been very vocal in their doctor bashing. Montana Senator Conrad Burns told a group of us last year that his blood boils at the thought of the power held by these congressional aides, most of whom, he said, never held a real job in their lives and never had to meet a payroll. "Elitists," he complained, "are deciding on your lives without knowing anything about them."

I don't plan to go into any real detail about the expected health proposal because we are very fortunate to have a very knowledgeable and articulate AMA Trustee with us from New Jersey, Dr. Pam Formica, who will speak later on the subject.

As we know, the Clinton health reform plan will certainly feature as its cornerstone "managed competition" which has the potential to evolve very quickly into an oligopoly in which a few large plans dominate the market, thereby limiting the competition "managed competition" advocates supposedly promote—something you can bet has not escaped the attention of the insurance monoliths who financially backed the Jackson Hole Group.

The Clinton health reform plan reportedly will include tort reform and some mechanisms to cut unnecessary bureaucracy, paperwork and hassles on physicians. There is even a rumor that the CLIA and OSHA regulations may be dumped. But will it really happen?

Managed competition may not alter those hassles. As Professor Enthoven himself has said: "Managed competition is not deregulation. It is new rules, not no rules." Unless changed by policy wonks, managed competition appears to put too much power into the hands of a nonmedical management intermediary whose primary goal and bottom line is to be economically viable.

As you probably know, Ira Magaziner, the White House's senior health policy adviser, claims that physicians will like the plan. I hope that he is right but it appears that all roads seem to lead to the rationing of care—with physicians placed in the untenable position of withholding the care and then being blamed for it.

Many more are beginning to understand that the whole idea of managed competition, as Uwe Reinhardt puts it, is to delegate the painful rationing decisions into the dark corners of the HMO—a smart way of moving them from the government to the private sector. But I must also say that it was Mr. Magaziner who recently said: "We need to get the lawyers out of the doctor-patient relationship."

I think that if the Administration were being consistent, however, what with a lawyer heading up the task force on health-care reform, President Clinton should appoint a physician to chair a national task force on tort reform. The task force would, of course, exclude the American Bar Association and the American Trial Lawyers Association.

One final comment on tort reform. We are all aware of the comments coming from the bureaucrats in Washington that 25% of the tests and x-rays performed are unnecessary. That statement came from a speech from former HHS Secretary Dr. Louis Sullivan.

What no one ever seems to mention is his next sentence in which he said that "many of these are ordered for defensive medicine purposes, and we need tort reform and practice parameters to eliminate the defensive medicine." We must remind everyone who will listen to us that a Harvard comprehensive study in 1991 revealed that 80% of all malpractice suits against physicians did not involve any negligence.

One of my greatest fears in this health care reform process is the seeming preoccupation of the Clinton task force in trying to find a generic substitute for physicians. The doctor bashers on the health panel are looking to limited licensed practitioners as some kind of panacea for rising health care costs. This slide towards "cheap" medicine, if you will, obviously could be devastating to both physician and patient.

There are indications that the Task Force will attempt to reduce some state limitations of limited license practitioners to clear the way for this strategy. The AMA, state and county medical societies must continue to be very vigilant in this regard. The word from the chiropractors in Arkansas is that Virginia Kelley, the president's mother, is a regular user of their services and they are hoping to parley that influence into generous insurance coverage for chiropractic.

We must remember that this is politics, and the mother's milk of politics is *quid pro quo*. No one subscribes to that more than Bill Clinton. Trade-offs will be a big part of his reform package and where the real action will be.

As presently planned, the Clinton health proposal will probably mean more work for primary care doctors and less for surgeons. Some hospitals will close. One thing for certain: the coming reform will have a far greater impact on jobs, corporate fortunes, and economic growth than even the much ballyhooed reduction in our military complex. About 10.6 million Americans, one out of every 11 in the work force, are employed in the health field.

Some of the numbers are surprising. In Pittsburgh, the Steel City, health care employs 106,000 people compared to only 25,000 in steel. Some 110,000 work in health care in the greater Houston area compared to only 66,000 in oil and gas. It is estimated that if medicine's current 14.4%

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

rate of GDP stops growing and shrinks back to 10%, the result could be a loss of millions of jobs in the health care field.

While many Clinton advisers are hoping that the plan will be another Social Security, others are fearing that it may be another Medicare Catastrophic Coverage Act of 1988 - the one that embarrassed the Congress and was repealed the next year.

As for the expected mandated or voluntary price controls in the Clinton plan, they simply have a very poor history of never working.

As said by the chairman of the ill-fated "Price Commission for Phase II of the wage-price controls under the Nixon Administration, C. Jackson Grayson, Jr., "we started Phase II with three and one half pages of regulations and ended with 1534. In an effort to correct one inequity, you create another."

Barry Bosworth, the former director of the Council of Wage and Price Stability during the Carter Administration, was quoted as saying that: "When we actually did it, you just never would have believed so many things could go wrong." But as Al Capone used to say: "A kind word will go a long way, but a kind word and a gun will go even farther".

Even Alain Enthoven opposes a global budget, and has warned members of Congress that it is akin to bombing from 35,000 feet: "from high altitudes in your Washington offices", he told them, "you do not see the faces of the people you are killing". It was poet William Blake who said that "suffering has a human face", and it is you physicians who see those faces and who will be treating that suffering—not President Clinton or the Congress. A cap on expenditures might actually work, if only we could place a cap on illness—but it doesn't work that way.

New York Senator Daniel Moynihan recently told *Business Week* that the overriding principle of health care reform should be: "First, do no harm". The publication said that the goal in reform is to get a grip on cost, extend coverage, and provide incentives for savings. That does not translate, it said, into a popular mandate to redo the whole health system.

Despite the strong public opinion in support of health reform, President Clinton himself doesn't have the power he had a few months ago.

Having won on their Stimulus Plan filibuster in the Senate, Republicans have a new vitality and will not roll over on health reform. They intend to introduce their own proposal along with House Republicans, possibly even before the Clinton plan sees the light of day.

Republican Senator Phil Gramm, and a former professor of economics, said that a health insurance purchasing cooperative "was like a five-legged animal; it may work, but we don't see any of them walking around."

Unless the plan substantially covers the elderly for long-term care, something that may be too difficult to achieve because of the high price tag, the AARP shows signs of not supporting health reform.

The reform package will have to work its way through six standing committees in the House and another four in the Senate, not to mention all the caucuses and conference committees which will follow that process.

But this is not to say that health care reform is unnecessary. It should be emphasized that physicians realize that we can not forever sustain the present growth of health care costs. But effective cost containment must take into account advancing technology, our aging population, physician manpower and specialty distribution, medical education, and research costs.

It should move long-term health care costs into our social service budget by acknowledging that our health care system is the catch-all for many societal problems: drug addiction, violence, poverty, and abusive life styles, to name just a few.

To curb costs, physicians support practice parameters, technology assessment, outcomes research, tort reform and the reduction of administrative hassles and paperwork - all of which would serve to reduce that growth and help the system operate more efficiently. All Americans must be insured and have appropriate access to the system.

Having said that, I should also say that physicians around the country are every bit as worried as you are as to what will really come out from the Task Force. Physicians who are discouraged from what they hear out of Washington must take heart because reform is not a done deal and can be influenced by physicians—as long as physicians stay together and don't fight each other. Even some of the doctor bashers acknowledge that the reform won't work without the cooperation of physicians.

With all the criticisms of you and your colleagues flowing freely from the uninformed and unknowing, there is a story that while it may not serve as a consolation to you, it certainly is appropriate for these trying times.

It involves a rather famous entertainer who was traveling back from a two-week public relations trip to China. On the same boat was a missionary who had worked with the poor in China for many years. When they docked in New York, the missionary saw a big crowd waiting at the pier—all of them fans of the entertainer.

"Lord, I don't understand," the missionary said. "I gave 42 years of my life to helping people, and he gave only two weeks—yet there are thousands welcoming him home and nobody here to welcome me." And the Lord replied, "Son, you're not home yet".

Since so much about health reform involves politics, perhaps it is appropriate to close on that note. As we all know, many people take their politics very seriously—In fact, all of Abraham Lincoln's relatives on his father's side and all except one on his mother's side voted against him for President simply because *they* were Democrats and *he* was a Republican.

That leads me to the story of the man, 90 years old who happened to be a staunch Republican all his life, who was experiencing failing health. Upon hearing from his physician that the end was near, he asked his son to take him to the courthouse so that he could change his voter registration from Republican to Democrat.

The young man protested vigorously, reminding his father of all the leadership positions he had held in the Republican party and his strong party feelings. Finally the old man replied to his son's scolding: "Son, if someone has to die, it might as well be one of them".

TACKLE THE ISSUES

HEALTH CARE REFORM

There is no better time for clinicians to pursue an advanced graduate degree in health care and management. New York University's Advanced Management Program for Clinicians (AMPC) offers an M.S. in management for physicians, nurses, social workers, and other health care professionals. Expand your background in health services management, financial management, and health policy to advance in your health care career and to make a difference in the health policy arena.

ROBERT F. Wagner
GRADUATE SCHOOL OF PUBLIC SERVICE

For more information, call (212) 998-7460 or write to Health Policy and Management Program, Robert F. Wagner Graduate School of Public Service, New York University, 40 West Fourth Street, Room 600, New York, N.Y. 10012-1118.

NEW YORK UNIVERSITY
A PRIVATE UNIVERSITY IN THE PUBLIC SERVICE

New York University is an affirmative action/equal opportunity institution.

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Mehdi S. Eslami, M.D., Delegate to AMA, and Guy W. Van Syckle, M.D., Past President, CSMS.

Edward Kamens, Chairman, CSMS AMA Delegation, and Palma E. Formica, M.D., Member AMA Board of Trustees.

9642

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

NOT TO CIRCULATE

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

HEALTH SCIENCES LIBRARY
UNIVERSITY OF MARYLAND AT
BALTIMORE

NOT TO CIRCULATE

WERT
BOOKBINDING
Grantville, Pa.
MAY - JUNE 1995
We're Quality Bound